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104

# Chemical Warfare in Southeast Asia and Afghanistan

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Report to the Congress  
from Secretary of State  
Alexander M. Haig, Jr.,  
March 22, 1982

THE SECRETARY OF STATE  
WASHINGTON

TO THE CONGRESS OF THE UNITED STATES:

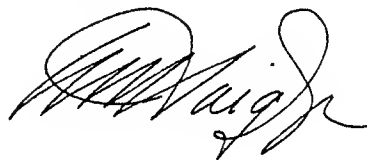
The years from 1914 to 1918 were among the most destructive of human life in mankind's history. Yet the sacrifice of millions brought no lasting peace. Of the elaborate structure for collective security, and the series of pacts outlawing war and controlling armaments which were negotiated in the aftermath of this First World War, little remains today. The League of Nations, the Kellogg-Briand Pact, and the Washington Naval Agreement were all swept away in the tide of aggression which culminated in a second global conflict. Almost the sole surviving monument, in the law of nations, to the twenty million dead of the First World War is the 1925 Geneva Protocol outlawing chemical and biological warfare.

Today this accord, among the oldest of arms control agreements still in force, along with another more recent such agreement banning biological and toxin weapons, is again in danger of being swept away by a new tide of aggression. Over the past seven years chemical and toxin weapons have been used, on an ever-widening scale, in genocidal campaigns against defenseless peoples. These weapons are being used for precisely the reason mankind has condemned and sought to outlaw them—because of their indiscriminate action and horrific effects. Today evidence of chemical and toxin warfare has accumulated to the point where the international community can no longer ignore the challenge.

The enclosed report on the use of chemical and toxin weapons by the Soviet Union and its Allies in Laos, Kampuchea, and Afghanistan has been prepared for submission to the Congress, to the United Nations, and to each member of the international community. The report is drawn from information made available to the United States Government since 1975. It contains the most comprehensive compilation of material on this subject available, and presents conclusions which are fully shared by all relevant agencies of the United States Government.

The international community and the world public need not rely solely on this report to form their judgment, nor only upon the United States to provide their information. Lethal chemical and toxin weapons are regrettably still in use in Laos, Kampuchea, and Afghanistan. New victims appear, new witnesses come forward, new scientific evidence is uncovered with increasing frequency. The great bulk of the information in the enclosed report could have been collected and analyzed by any interested government, international organization, or major news service. If the efforts of the United States Government to call attention to chemical warfare in Afghanistan and Southeast Asia stimulate others to discover for themselves, and join in efforts to expose the truth, this report will have served its most important purpose.

Sincerely,

A handwritten signature in dark ink, appearing to read 'A. Haig, Jr.', with a stylized, cursive script.

Alexander M. Haig, Jr.

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*This study presents the evidence available to the U.S. Government on chemical warfare activities in Laos, Kampuchea, and Afghanistan through January 1982 and examines the Soviet involvement in those activities. It is based on a massive amount of information, from a variety of sources, which has been carefully compiled and analyzed over the years. The paper is accompanied by annexes and tables that provide details of the medical evidence and sample analyses, a technical description of trichothecene toxins, and other supporting data.*

## INTRODUCTION

Nearly 7 years ago, reports of the use of lethal chemical weapons began to emerge from Laos. In 1978, similar reports started to come from Kampuchea, and in 1979 from Afghanistan. Early reports were infrequent and fragmentary, reflecting the remoteness of the scene of conflict and the isolation of those subjected to such attacks. In the summer of 1979, however, the State Department prepared a detailed compilation of interviews with refugees from Laos on this subject. That fall, a U.S. Army medical team visited Thailand to conduct further interviews. By the winter of 1979, the United States felt that it had sufficiently firm evidence of chemical warfare to raise the matter with the governments of Laos, Vietnam, and the Soviet Union. All three governments denied that a basis for concern over the use of chemical warfare agents existed.

Dissatisfied with these responses, and possessing further reports that lethal chemical agents were in use in Southeast Asia and Afghanistan, the U.S. Government in 1980 began to raise the issue publicly in the United Nations, with the Congress, and in other forums. In August of that year, the State Department provided extensive documentation containing evidence of chemical weapons attacks to the United Nations and also made this material publicly available. In December, as a result of efforts by the United States and other concerned nations, the U.N. General Assembly voted to initiate an international investigation into the use of chemical weapons. This investigation is still underway. To date, the U.N. investigating team has been denied admission to any of the three countries where these weapons are in use.

Despite the volume of information on chemical warfare in Southeast Asia which had become available by 1980,

there remained one major unresolved issue—the exact nature of the chemical agents in use. Collection of physical samples was hindered by the remoteness of the then principal areas of conflict—as many as 6 weeks by foot to the nearest international border. Tests for known chemical warfare agents on those samples that were obtained proved consistently negative.

In order to identify the chemical agents in use, U.S. experts in late 1980 began to go back over all the reporting—as far back as 1975—looking for new clues. In particular, they sought to match the reported symptomatology of victims—which commonly included skin irritation, dizziness, nausea, bloody vomiting and diarrhea, and internal hemorrhaging—with possible causes. As a result of this review, the U.S. Government in mid-1981 began to test physical samples from Southeast Asia for the presence of toxins. These substances are essentially biologically produced chemical poisons. Although they have never before been used in war, this was a technical possibility, and it was noted that certain toxins could produce the sorts of symptoms observed in Southeast Asian victims of chemical warfare.

In August 1981, unnatural levels and combinations of lethal trichothecene toxins were detected in the first sample to be tested by the United States for such agents. This consisted of vegetation taken from a village in Kampuchea where an attack occurred in which people had died after exhibiting the symptoms described above. In succeeding months, further samples, taken from the sites of attacks in both Kampuchea and Laos, yielded similar results. So did samples of blood taken from victims of a chemical attack in Kampuchea.

Despite a continued flow of reports, dating back over 7 years, of chemical warfare in Southeast Asia and more recently Afghanistan, and despite the still mounting physical evidence of the use of trichothecene toxins as warfare agents, doubts as to the conclusive nature of the available evidence have persisted. These doubts have arisen for several reasons. For one, the evidence of the use of lethal chemical weapons has become available over a period of several years and from a variety of sources. Few governments, journalists, or interested members of the public have been exposed to all of this evidence, nor has it been available in any one place. A second difficulty has been the inevitable need for the U.S. Government to protect some of the relevant information, often gathered at personal risk to individuals who secured it, or obtained through the use of highly sensitive methods.

## Chronology of Diplomatic/International Actions on Chemical Warfare Use

### October 1978

The United States called to the attention of the Lao Charge d'Affaires in Washington the press reports alleging use of poison gas in Laos.

Assistant Secretary of State for East Asian and Pacific Affairs Holbrooke traveled to Vientiane and discussed our concerns over H'Mong human rights and other issues with Lao leaders.

### Late 1978

The Department of State directed U.S. diplomatic missions in the Southeast Asia area to seek to develop information on the alleged use of poison gas against the H'Mong.

### January 1979

The Department of State again informed the Lao Embassy of U.S. concerns about reports of poison gas use in Laos, coupling this with a similar demarche in Vientiane. The Lao denied the validity of the reports.

### March 1979

The U.S. Representative to the 35th session of the U.N. Human Rights Commission expressed U.S. concern about the plight of the H'Mong, specifically raising the poison gas use issue.

### May 1979

A State Department representative went to refugee camps in Thailand to interview H'Mong claiming to be eyewitnesses and/or victims of poison gas attacks in Laos.

A State Department representative visited Vientiane where he discussed the problem with various diplomatic missions and the senior U.N. representative in Laos. During that visit, he raised U.S. concerns about the problem directly with the Lao Foreign Ministry.

### September 1979

A Department of Defense medical team was dispatched to Thailand to interview and prepare a report on H'Mong refugees having knowledge of gas attacks in Laos.

### November 1979

Demarches were made to the Vietnamese in Paris and to the Soviets in Moscow expressing U.S. concerns about reports of poison gas being used against "resistance forces" in Laos. Both the Soviets and Vietnamese supported the Lao denial of the validity of the reports.

### December 1979

State and Defense Department officials presented evidence of gas attacks in Laos to the House Foreign Affairs Committee.

**February 1980**

A bilateral demarche was made to the Soviets about U.S. concerns regarding chemical warfare use in both Laos and Kampuchea and about reports that chemical weapons were being used by the Soviets in Afghanistan. The demarche was made in Geneva in the context of the U.S./Soviet bilateral negotiations on a comprehensive prohibition of chemical weapons production, development, and stockpiling.

**May 1980**

An interagency team of U.S. Government political, technical, and intelligence officers was dispatched to Europe to brief the allies about the problem and to stimulate support for having an impartial international investigation conducted.

**July 1980**

Another bilateral demarche was made to the Soviets in the context of the U.S./Soviet bilateral chemical warfare negotiations, concerning the problem of the reported use of chemical weapons in both Southeast Asia and Afghanistan.

The Inter-Parliamentary Union adopted a resolution calling for an impartial international investigation of reports of chemical weapons use.

**August 1980**

The United States circulated to U.N. member states a 125-page compendium of reports and declassified intelligence information pertaining to the use of chemical weapons in Laos, Kampuchea, and Afghanistan.

The 40-nation Committee on Disarmament included language in its Annual Report to the U.N. General Assembly on the need for an impartial international investigation of the problem of chemical weapons use.

**December 1980**

With the full and active support of the United States, the West, and others, the U.N. General Assembly adopted a resolution (A/35/144 C) establishing a U.N. investigation, under the auspices of the U.N. Secretary General and with the assistance of qualified medical and technical experts, of reports of chemical weapons use. The vote was 78 in favor to 17 opposed, with 36 abstentions.

**March 1981**

In accordance with U.N. General Assembly Resolution A/35/144 C and the request of the U.N. Secretary General, the U.S. submitted detailed information pertaining to the reports of the use of chemical weapons in Southeast Asia and Afghanistan. The U.S. submission consisted of a letter summarizing

the U.S. submission, the U.S. compendium of reports from August 1980, an update to that compendium covering the period through January-February 1981, the transcripts of congressional hearings held on the subject in December 1979 and in April 1980, and the texts of House and Senate resolutions condemning the use of chemical weapons.

**July 1981**

The United States provided further details and written responses to questions from the U.N. Group of Experts concerning the U.S. submission of March 1981.

**September 1981**

Secretary Haig announced, in his September 13 speech in Berlin, that the United States had obtained physical evidence of the use of lethal mycotoxins in Southeast Asia, discovered in the analysis of a leaf and stem sample obtained from the site of a chemical attack in Kampuchea.

On September 14, the United States submitted a report on the new evidence pertaining to the use of mycotoxins to the U.N. Group of Experts investigating reports of chemical weapons use.

Under Secretary of State for Political Affairs Stocssel held a press conference in Washington on September 14 and provided a detailed press backgrounder on the new evidence.

Secretary Haig raised U.S. concerns about the new evidence pertaining to the use of lethal mycotoxins in Southeast Asia and about the 1979 Sverdlovsk anthrax incident with Soviet Foreign Minister Gromyko during their bilateral consultations at the United Nations in New York.

**October 1981**

Following up the Haig/Gromyko discussions, detailed bilateral demarches were made to the Soviets in Washington by Acting Arms Control and Disarmament Agency Director Grey, and a followup in Moscow by the U.S. Deputy Chief of Mission, on the general subject of Soviet Biological Warfare Convention compliance and specific U.S. concerns regarding the 1979 Sverdlovsk anthrax incident and the evidence of the use of trichothecene mycotoxins in Southeast Asia. The Soviets rejected U.S. concerns once again in their formal response in November.

An interagency team of political, technical, and intelligence officers was dispatched to Europe to brief the allies about the new evidence of the use of lethal mycotoxins in Southeast Asia.

A delegation of U.S. Government political, technical, and medical experts appeared

before the U.N. Group of Experts to respond to questions pertaining to the U.S. submission on September 14 of new evidence concerning the use of lethal mycotoxins in Southeast Asia.

**November 1981**

The U.N. Group of Experts investigating reports of chemical weapons use traveled to Thailand to visit refugee camps and interview and examine survivors and eyewitnesses of chemical attacks in Laos and Kampuchea. While there, the experts also obtained samples from alleged chemical attacks and samples of vegetation and blood from refugees exposed to chemical attacks.

Richard Burt, Director of the Bureau of Politico-Military Affairs, in testimony before the Congress, announced the results of analyses of additional samples of chemical warfare use revealing the presence of high levels of mycotoxins and the results of analyses of control samples from Southeast Asia which were found to contain no mycotoxins.

The United States submitted a report on its analyses of chemical warfare use samples from both Kampuchea and Laos, which were found to contain high levels of mycotoxins, to the U.N. Group of Experts investigating reports of chemical weapons use.

Demarches were made to the Vietnamese in New York and to the Lao in Vientiane regarding the evidence of the use of lethal mycotoxins in the conflicts in Kampuchea and Laos. Both the Vietnamese and the Lao rejected the evidence and denied the validity of U.S. concerns.

**December 1981**

The U.N. Secretary General submitted the Report of the U.N. Group of Experts investigating reports of chemical weapons use (A/36/613). The report was inconclusive and stated that the group had been unable to carry out all the actions it had intended (i.e., on-site visits to Afghanistan, Laos, and Kampuchea) due to the refusals to cooperate of the countries concerned, and that it had been unable to complete some of the actions it had planned (e.g., on-site visits to Pakistan, analysis of the samples obtained in Thailand) in the time available.

With the full and active support of the United States, the West, and others, the U.N. General Assembly adopted a resolution (A/36/96 C) extending for another year the mandate of the U.N. Secretary General's Group of Experts investigating reports of chemical weapons use. The vote on the resolution was 86 in favor to 20 opposed, with 32 abstentions.

This report represents an effort of the U.S. Government to correct the first deficiency and to ameliorate the second to the extent possible. In preparation of this report, all of the information available to the U.S. Government on chemical weapons use in Laos, Kampuchea, and Afghanistan was assembled in one place. This information was again reviewed, analyzed, cross-indexed, and organized in a coherent fashion. Based upon this comprehensive analysis, a set of conclusions were drawn, conclusions which have since been reviewed and agreed on without qualification by every relevant agency of the U.S. Government.

The evidence upon which this report is based is of several kinds, including:

- Testimony of those who saw, experienced, and suffered from chemical weapons attacks;
- Testimony of doctors, refugee workers, journalists, and others who had the opportunity to question large numbers of those with firsthand experience of chemical warfare;
- Testimony of those who engaged in chemical warfare or were in a position to observe those who did;
- Scientific evidence, based upon the analysis of physical samples taken from sites where attacks had been conducted;
- Documentary evidence from open sources; and
- Intelligence derived from "national technical means."

These sources provide compelling evidence that tens of thousands of unsophisticated and defenseless peoples have for a period of years been subjected to a campaign of chemical attacks. *Taken together, this evidence has led the U.S. Government to conclude that Lao and Vietnamese forces, operating under Soviet supervision, have, since 1975, employed lethal chemical and toxin weapons in Laos; that Vietnamese forces have, since 1978, used lethal chemical and toxin agents in Kampuchea; and that Soviet forces have used a variety of lethal chemical warfare agents, including nerve gases, in Afghanistan since the Soviet invasion of that country in 1979.*

The implications of chemical warfare in Afghanistan and Southeast Asia are painful to contemplate but dangerous to ignore. This activity threatens not only the peoples of those isolated regions but the international order upon which the security of all depends. Those who today suffer chemical warfare against their homelands are powerless to stop it. The prohibitions of international law and solemn agreement are not self-enforcing.

Only an alert and outspoken world community, intent to maintain those standards of international behavior it has so painfully achieved and so tenuously established, can bring sufficient pressure to bear to halt these violations of law and treaty. It is hoped that publication of this report will be one step in this process, the end result of which will be the cessation of chemical warfare and the strengthening of the rule of law in the affairs of nations.

## KEY JUDGMENTS

**Laos.** The U.S. Government has concluded from all the evidence that selected Lao and Vietnamese forces, under direct Soviet supervision, have employed lethal trichothecene toxins and other combinations of chemical agents against H'Mong resisting government control and their villages since at least 1976. Trichothecene toxins have been positively identified, but medical symptoms indicate that irritants, incapacitants, and nerve agents also have been employed. Thousands have been killed or severely injured. Thousands also have been driven from their homeland by the use of these agents.

**Kampuchea.** Vietnamese forces have used lethal trichothecene toxins on Democratic Kampuchean (DK) troops and Khmer villages since at least 1978. Medical evidence indicates that irritants, incapacitants, and nerve agents also have been used.

**Afghanistan.** Soviet forces in Afghanistan have used a variety of lethal and nonlethal chemical agents on *mujahidin* resistance forces and Afghan villages since the Soviet invasion in December 1979. In addition, there is some evidence that Afghan Government forces may have used Soviet-supplied chemical weapons against the *mujahidin* even before the Soviet invasion. Although it has not been possible to verify through sample analysis the specific agents used by the Soviets, a number of Afghan military defectors have named the agents brought into the country by the Soviets and have described where and when they were employed. This information has been correlated with other evidence, including the reported symptoms, leading to the conclusion that nerve agents, phosgene oxime, and various incapacitants and irritants have been used. Other agents and toxic smokes also are in the country. Some reported symptoms are consistent with those produced by lethal or

sublethal doses of trichothecene toxins, but this evidence is not conclusive.

**The Soviet Connection.** The conclusion is inescapable that the toxins and other chemical warfare agents were developed in the Soviet Union, provided to the Lao and Vietnamese either directly or through the transfer of know-how, and weaponized with Soviet assistance in Laos, Vietnam, and Kampuchea. Soviet military forces are known to store agents in bulk and move them to the field for munitions fill as needed. This practice also is followed in Southeast Asia and Afghanistan, as evidenced by many reports which specify that Soviet technicians supervise the shipment, storage, filling, and loading onto aircraft of the chemical munitions. The dissemination techniques reported and observed evidently have been drawn from years of Soviet chemical warfare testing and experimentation. *There is no evidence to support any alternative explanation, such as the hypothesis that the Vietnamese produce and employ toxin weapons completely on their own.*

## METHODOLOGY

The judgments of this study were arrived at through a rigorous analytical process.

- Every relevant piece of information on reported chemical warfare incidents was reviewed, recorded, and tabulated. Numbers of attacks and deaths were screened for possible duplication. Extensive data on the Soviet chemical and biological warfare program also were reviewed.

- All the test data on physical evidence available to the U.S. Government—including environmental samples and background controls—were reviewed.

- A scientific report on toxins, which concluded that trichothecenes probably were among the agents used in Southeast Asia, was prepared.

- The medical evidence was analyzed, drawing on all available information from Southeast Asia and Afghanistan and incorporating the findings of a Department of Defense medical team, which concluded that at least three types of agents were used in Laos.

- Extensive consultations were held with government and nongovernment scientists and medical authorities, many of whom were asked to review the evidence. Experts from other countries also were consulted.

After the data were organized to permit comparative analysis, the study focused on three separate questions.

- Have lethal and other casualty-producing agents been used in Southeast Asia and Afghanistan?
- What are these agents, and how and by whom are they employed?
- Where do these agents originate, and how do they find their way to the field?

Although the evidence differs for each country, the analytical approach was the same. Testimony of eyewitnesses—date, place, and type of attack—was matched against information from defectors, journalists, international organizations, and sensitive information that often pinpointed the time and place of chemical attacks. In addition, information on military operations in the areas where chemical attacks had been reported was examined to establish whether air or artillery strikes took place or whether there was fighting in the areas where chemical agents reportedly were used. *In all three countries, instances were identified in which eyewitness accounts could be correlated directly with information from other sources on military operations in progress.*

There is no evidence of any systematic propaganda campaign by either the H'Mong in Laos or the Afghan resistance forces to promote the allegation that chemical agents have been used on their people. On the other hand, there were early indications that Pol Pot's Democratic Kampuchean resistance did engage in an organized propaganda campaign on chemical agent use. These indications made U.S. Government analysts cautious about accepting DK allegations, which increased markedly after the chemical attacks in Laos were publicized. For Kampuchea, therefore, special efforts were taken to confirm such allegations by analyzing sources of information that in no way could be considered part of a propaganda or deception campaign.

## DISCUSSION OF FINDINGS

In September 1981, the U.S. Government declared publicly that toxins—poisonous chemical substances extracted from biological material—probably were the mysterious lethal agents used for many years in Laos and Kampuchea. The statement was prompted by the discovery of high levels of trichothecene toxins in a vegetation sample collected shortly after a March 1981 Vietnamese chemical attack in Kampuchea. This con-

clusion, however, rested on a much broader base of evidence than analysis of one sample.

By April 1980, the U.S. Government had already concluded that lethal agents almost certainly had been used against H'Mong tribespeople in Laos. There was less certainty then about the use of lethal agents in Kampuchea, mainly because of the already mentioned suspicions about the propaganda campaign of Pol Pot's Democratic Kampuchean forces, although their claims subsequently were shown to be valid. It was also concluded that chances were about even that lethal agents had been used in Afghanistan. There was little doubt by April 1980 that riot-control agents and some form of incapacitants had been used in all three countries. Since that April 1980 assessment, additional evidence has allowed a much firmer conclusion. There is now no doubt that casualties and deaths have resulted from chemical attacks in all three countries.

### What Chemical Agents Are Being Used?

As soon as it was determined that chemical agents had been used, an effort was made to identify the specific agents. To do this it was necessary to collect and analyze at least one of the following: environmental samples contaminated with agents, the munitions used to deliver agents, or biological specimens from victims of an attack. A study by medical-toxicological experts of symptoms exhibited by individuals exposed to toxic agents provides a good indication of the general class of chemical agent used. Thus, the range of clinical manifestations from chemical agents, as reported by a U.S. Army investigative team in Thailand, resulted in the determination that nerve agents, irritants such as CS, and highly toxic hemorrhagic chemicals or mixture of chemicals were used in Laos.

Other medical-toxicological personnel who reviewed the evidence and conducted their own investigation reached the same conclusion. They further indicated that toxins such as the trichothecenes were a probable cause of the lethal hemorrhaging effect seen in Kampuchea and Laos. In many cases, symptoms reported by the Democratic Kampuchean forces in Kampuchea and the *mujahidin* in Afghanistan were similar to those reported by the H'Mong in Laos. Moreover, symptoms reported from Afghanistan and Kampuchea indicated that a highly potent, rapid-acting, incapacitant "knockout" chemical also was being used. *Mujahidin* victims and wit-

nesses to chemical attacks reported other unusual symptoms, including a blackening of the skin, severe skin irritation along with multiple small blisters and severe itching, severe eye irritation, and difficulty in breathing—all of which suggests that phosgene oxime or a similar substance was used.

Collecting samples possibly contaminated with a toxic agent during or after a chemical assault is difficult under any circumstances but particularly when the assault is against ill-prepared people without masks or other protective equipment. Obtaining contaminated samples that will yield positive traces of specific chemical agents depends on many factors. These include the persistency of the chemical, the ambient temperature, rainfall, wind conditions, the medium on which the chemical was deposited, and the time, care, and packaging of the sample from collection to laboratory analysis.

Many traditional or known chemical warfare agents are nonpersistent and disappear from the environment within a few minutes to several hours after being dispersed. Such agents include the nerve agents sarin and tabun; the blood agents hydrogen cyanide and cyanogen chloride; the choking agents phosgene and diphosgene; and the irritant phosgene oxime. Other standard chemical warfare agents—such as the nerve agents VX and thickened soman and the blistering agents sulfur mustard, nitrogen mustard, and lewisite—may persist for several days to weeks depending on weather conditions.

The trichothecene toxins have good persistency but may be diluted by adverse weather conditions to below detectable concentrations. To maximize the chances of detection, sample collections need to be made as rapidly as possible after a chemical assault; as with many agents, this means minutes to hours. Under the circumstances of Southeast Asia and Afghanistan, such rapid collection has simply not been possible. Although many samples were collected, few held any realistic prospect of yielding positive results. It is fortunate that trichothecenes are sufficiently persistent and in some cases were not diluted by adverse weather conditions. Thus we were able to detect them several months after the attack.

Samples have been collected from Southeast Asia since mid-1979 and from Afghanistan since May 1980. To date, about 50 individual samples—of greatly varying types and usefulness for analytical purposes—have been collected and analyzed for the presence of known

chemical warfare agents, none of which has been detected. Based on recommendations by medical and toxicological experts and findings of investigators from the U.S. Army's Chemical Systems Laboratory, several of the samples have been analyzed for the trichothecene group of mycotoxins. Four samples, two from Kampuchea and two from Laos, were found to contain high levels of trichothecene toxins. In addition, preliminary results of the analysis of blood samples drawn from victims of an attack indicate the presence of a trichothecene metabolite of T-2, namely HT-2.

A review of all reports indicates the use of many different chemical agents, means of delivery, and types of chemical attacks. The use of trichothecene toxins has been identified through symptoms and sample analysis. In some cases, however, the symptoms suggest other agents, such as nerve gas, which have not been identified through sample analysis. Significant differences as well as similarities have surfaced in the reports from the three countries. The evidence from each country, therefore, is described separately, with attention drawn to similarities where appropriate.

## Laos

Reports of chemical attacks against H'Mong villages and guerrilla strongholds in Laos date from the summer of 1975 to the present (see Table 1). Most of the reports were provided by H'Mong refugees who were interviewed in Thailand and the United States. More than 200 interviews were carried out variously by U.S. Embassy officials in Thailand, a Department of Defense team of medical-toxicological experts (see Annex B), U.S. physicians, Thai officials, journalists, and representatives of international aid and relief organizations. According to the interviews, Soviet AN-2 and captured U.S. L-19 and T-28/41 aircraft usually were employed to disseminate toxic chemical agents by sprays, rockets, and bombs. In some cases, Soviet helicopters and jet aircraft were said to have been used.

The reports describe 261 separate attacks in which at least 6,504 deaths were cited as having resulted directly from exposure to chemical agents. The actual number of deaths is almost certainly much higher, since the above figure does not take account of deaths in attacks for which no specific casualty figures were reported. The greatest concentration of reported chemical agent use occurred in the area where the three

TABLE 1

### Laos: Summary of Reported Chemical Attacks and Associated Deaths, 1975-81

| Time Period    | Area        | Attacks <sup>a</sup> | Deaths <sup>b</sup> |
|----------------|-------------|----------------------|---------------------|
| Summer 1975    | Vientiane   | 2                    | 25 +                |
| Fall 1976      | Phou Bia    | 8                    | 10                  |
|                | Savannakhet | 1                    | 10                  |
| Winter 1976-77 | Phou Bia    | 2                    | 16                  |
| Spring 1977    | Phou Bia    | 6                    | 66 +                |
|                | Khammouan   | 2                    | 1                   |
| Summer 1977    | Phou Bia    | 6                    | 95                  |
| Fall 1977      | Phou Bia    | 1                    | 25                  |
| Winter 1977-78 | Phou Bia    | 10                   | 1,328 +             |
|                | Savannakhet | 6                    | 224                 |
| Spring 1978    | Phou Bia    | 34                   | 969 +               |
| Summer 1978    | Phou Bia    | 22                   | 664 +               |
| Fall 1978      | Phou Bia    | 19                   | 572                 |
| Winter 1978-79 | Phou Bia    | 5                    | 15 +                |
| Spring 1979    | Phou Bia    | 36                   | 257 +               |
| Summer 1979    | Phou Bia    | 5                    | 239 +               |
| Fall 1979      | Phou Bia    | 10                   | 56                  |
|                | Xaignabouri | 2                    | 24 +                |
|                | Phou Bia    | 4                    | 10 +                |
| Winter 1979-80 | Phou Bia    | 3                    | 24                  |
| Spring 1980    | Phou Bia    | 6                    | 187 +               |
| Summer 1980    | Phou Bia    | 1                    | 12                  |
|                | Xaignabouri | 7                    | 88 +                |
|                | Savannakhet | 3                    | 1 +                 |
| Winter 1980-81 | Xaignabouri | 2                    | 57                  |
|                | Phou Bia    | 4                    | 82                  |
|                | Vientiane   | 1                    | 1 +                 |
| Spring 1981    | Houaphan    | 2                    | ?                   |
|                | Phou Bia    | 7                    | 218                 |
|                | Vientiane   | 1                    | —                   |
| Summer 1981    | Phou Bia    | 1                    | ?                   |
| Fall 1981      | Phou Bia    | 4                    | 500 +               |
|                | Khammouan   | 3                    | 534 +               |
|                |             | 226                  | 6,310 +             |

<sup>a</sup> This tabulation omits 35 attack sites, accounting for 194 deaths, which could not be geographically located in the reports. The totals overall were 261 attacks and more than 6,504 deaths.

<sup>b</sup> A plus sign indicates that the report(s) of deaths gave a minimum figure. In some cases (shown with a question mark) deaths were reported, but no number was given. Other reports (signified with a dash) gave no information on fatalities.

provinces of Vientiane, Xiangkhoang, and Louangphrabang adjoin (see map). This triborder region accounted for 77% of the reported attacks and 83% of the chemical-associated deaths. Most of the reported attacks took place in 1978 and 1979. Since 1979, the incidence of chemical attacks appears to have been lower, but reported death rates among unprotected and untreated victims were higher. Only seven chemical attacks were reported in the fall of 1981, for example, yet 1,034 deaths were associated with those incidents.

The medical symptoms reportedly produced by the chemical agents are varied. According to knowledgeable physicians, the symptoms clearly point to at least three types of chemical agents—incapacitant/riot-control agents, a nerve agent, and an agent causing massive hemorrhaging. The last-named was positively identified as trichothecene toxins. This was announced publicly by Secretary Haig in September 1981.

In a number of the refugee reports, eyewitnesses described attacks as consisting of "red gas" or a "yellow cloud."

# Laos: Chemical Warfare Operational Areas



Red gas was considered the more lethal. A former Lao Army captain stated that the "red gas" caused the H'Mong to die within 12 hours. An employee of an international organization interviewed victims of a September 15, 1979 attack in which nonlethal rounds preceded an attack by five or six "red gas" bombs that covered a 500-meter area. Persons within 30-100 meters of the circle died in 10 minutes after severe convulsions. Others had headaches, chest pains, and vomiting but did not die.

Every qualified interrogator who systematically interviewed the H'Mong refugees concluded that they had been subjected to chemical attacks. A U.S. Government medical team returned from Thailand in 1979 convinced that several unidentified chemical warfare agents had produced the symptoms described by the refugees. This evidence was expanded by testimony from a variety of sources, including that of a Lao pilot who flew chemical warfare missions before defecting in 1979. His detailed description of the Lao, Vietnamese, and Soviet program to use chemical agents to defeat the H'Mong resistance helped dispel any lingering suspicions that the refugees had fabricated or embellished the stories. The Lao pilot described the chemical rocket he had fired as having a more loosely fitting warhead than a conventional rocket. (His account appears in Annex A.)

In 1977, a H'Mong resistance leader found a U.S. 2.75-inch rocket\* with a modified Soviet warhead that fits the Lao pilot's description. Other sources reported that U.S. 2.75-inch rockets were fitted with Soviet-supplied lethal chemical warheads by Soviet and Vietnamese technicians at facilities in Vientiane as well as in Xiangkhoang and Savannakhet Provinces. Munitions storage facilities suitable for storing chemical agents and weapons have been identified in each of these provinces. The aircraft types—AN-2s, L-19s, and T-28/41s—most often reported by the H'Mong refugees as being used to deliver chemical agents have been identified as based on airfields in northern Laos throughout this period. A special Lao Air Force unit is responsible for chemical rockets. The unit is commanded by a Soviet-trained Lao and has a Soviet rocket expert attached as an adviser.

\* During withdrawal of U.S. forces from Vietnam, thousands of these fell into Vietnamese hands.

Obtaining additional data for Laos has been difficult because of the nature of the fighting there. There have been few major operations. The reports reflect numerous minor engagements between the opposing forces. In nearly all cases, the chemical use reported has been directed against villages, in the absence of obvious combat operations. This lends support to the Lao pilot's claim that the Vietnamese and Lao military commands were engaged in a "H'Mong extermination" campaign.

Of particular interest are the circumstances surrounding the collection of two physical samples found to contain lethal toxins. The first was collected after a March 13, 1981 attack on a village between the villages of Muong Chai and Phakhao in the Phou Bia region. In this case, a large two-engine plane reportedly sprayed a mist of a moist, yellow, sticky substance; two villagers and all village animals died. The second sample is from Ban Thonghak, another village in the Phou Bia region, collected following an April 2, 1981 attack in which a jet aircraft reportedly sprayed a yellow substance; 24 of the 450 villagers died. In the spring of 1981, seven separate chemical attacks, resulting in 218 deaths, were reported to have occurred in this region.

It is significant that these attacks took place following a period of escalation in overall resistance activities in the Phou Bia area in the winter of 1980-81. During that period, joint suppression operations by Lao People's Liberation Army and Vietnamese Army forces had achieved only limited success, perhaps spurring both forces on to greater effort. The more intense use of chemical weapons may have been part of this effort.

Evidently the fact that chemical agents were being used in Laos was not widely known among units of the Lao Army. In June 1981, a group of refugees from a village in Vientiane Province reached Thailand and described attacks against them carried out a month earlier by helicopters "dropping poison" into their water supply. Lao field units subsequently entered the village and were surprised at the sight of many villagers still suffering from symptoms of acute poisoning. According to a villager, when the Lao military personnel saw the "small yellow grains" spread around the village, they were convinced that toxic chemicals had been used on the village and requested medical assistance for those villagers still suffering from nausea and bloody diarrhea.

In a December 15, 1981 press conference in Beijing, former Lao Health Ministry Bureau Director Khamsengkeo Sengsatit—who had defected to China—confirmed that chemical weapons were being used "in the air and on the ground" in Laos, killing "thousands." He asserted that the Vietnamese alone were using such weapons, keeping the matter secret from the Lao. He also stated that 3,000 Soviet advisers were in Laos and "have taken control" of the Lao Air Force, while 40,000-50,000 Vietnamese troops had "reduced Laos to the status of a colony."

### Kampuchea

Since October 1978, radio broadcasts, press releases, and official protests to the United Nations by the Democratic Kampuchea leadership have accused the Vietnamese and the Hanoi-backed People's Republic of Kampuchea regime of using Soviet-made lethal chemical agents and weapons against DK guerrilla forces and civilians. DK allegations for a time were the only source of information concerning chemical warfare attacks in Kampuchea. In November 1979, however, the guerrilla forces of the Khmer People's National Liberation Front reported that the Vietnamese had attacked them with a tear gas which, from their description, resembled the riot-control agent CS. Subsequently, Thai officials, Democratic Kampuchea informants and refugees, Vietnamese Army defectors, U.S. and Thai medical personnel, officials of international aid and relief organizations, and Canadian and West European officials also have implicated the Vietnamese in the offensive use of lethal and incapacitating chemical agents in Kampuchea.

There are reports of 124 separate attacks in Kampuchea from 1978 to the fall of 1981 in which lethal chemicals caused the deaths of 981 persons (see Table 2). The mortality figure represents a minimum because some reports state only that there were deaths and do not provide a number. The earliest reports cite attacks in Ratanakiri Province, in the northeastern corner of the country (see map). Reports from 1979 to the present show the use of lethal chemicals primarily in the provinces bordering Thailand. The greatest use of chemical agents apparently has been in Battambang Province, with 51 reported incidents; Pursat Province has experienced the next highest frequency, with 25

TABLE 2

**Kampuchea: Summary of Reported Chemical Attacks and Associated Deaths, 1978-81**

| Time Period    | Area              | Attacks | Deaths <sup>a</sup> |
|----------------|-------------------|---------|---------------------|
| 1978           | Ratanakiri        | 5       | ?                   |
| Summer 1979    | Kompong Speu      | 4       | 37                  |
| Fall 1979      | Siem Reap         | 1       | —                   |
|                | Battambang        | 4       | 22 +                |
|                | Pursat            | 2       | 1 +                 |
|                | Koh Kong          | 2       | 6 +                 |
|                | Kampot            | 1       | 3                   |
|                | Kompong Chhnang   | 2       | 118                 |
| Winter 1979-80 | Battambang        | 12      | 64 +                |
|                | Pursat            | 5       | 21 +                |
|                | Koh Kong          | 2       | 4                   |
| Spring 1980    | Battambang        | 3       | 20 +                |
|                | Pursat            | 8       | 24 +                |
|                | Koh Kong          | 5       | 13                  |
| Summer 1980    | Siem Reap         | 1       | 82 +                |
|                | Battambang        | 3       | 23 +                |
|                | Pursat            | 2       | 7                   |
|                | Koh Kong          | 3       | —                   |
| Winter 1980-81 | Battambang        | 8       | —                   |
|                | Pursat            | 2       | 3                   |
| Spring 1981    | Preah Vihear      | 1       | —                   |
|                | Battambang        | 12      | 163 +               |
|                | Pursat            | 3       | 42 +                |
|                | Koh Kong          | 1       | —                   |
|                | Kampot            | 1       | —                   |
| Summer 1981    | Battambang        | 3       | 7 +                 |
|                | Kompong Thom/Cham | 1       | —                   |
| Fall 1981      | Siem Reap         | 16      | 305                 |
|                | Battambang        | 6       | 16                  |
|                | Pursat            | 3       | —                   |
|                | Koh Kong          | 1       | —                   |
|                | Kampot            | 1       | —                   |
|                |                   | 124     | 981                 |

<sup>a</sup> A plus sign indicates that the report(s) of deaths gave a minimum figure. In some cases (shown with a question mark) deaths were reported, but no number was given. Other reports (signified with a dash) gave no information on fatalities.

reported incidents. These numbers are consistent with the overall high level of military activity reported in the border provinces.

A review of information from all sources provides direct and specific support for 28 of 124 reported attacks. There is, in addition, some evidence that in all reported instances some form of attack took place. This evidence includes reports of troop movements, supply transfers, operational plans, postoperation reporting, and air activity. It indicates that military activity took place at the time and place of every incident reported to involve lethal chemical agents. In some cases, it provides strong circumstantial evidence that the action

involved chemical substances—for example, the movement of chemicals and personal protection equipment into the area.

There is no doubt that in late 1978 and 1979 the Vietnamese, and what later became the People's Republic of Kampuchea forces, made at least limited use of riot-control chemicals and possible incapacitating agents against both Communist and non-Communist guerrilla forces in Kampuchea. The chemicals used probably included toxic smokes, riot-control agents such as CS, and an unidentified incapacitating agent that caused vertigo and nausea and ultimately rendered victims unconscious with no other signs or symptoms.

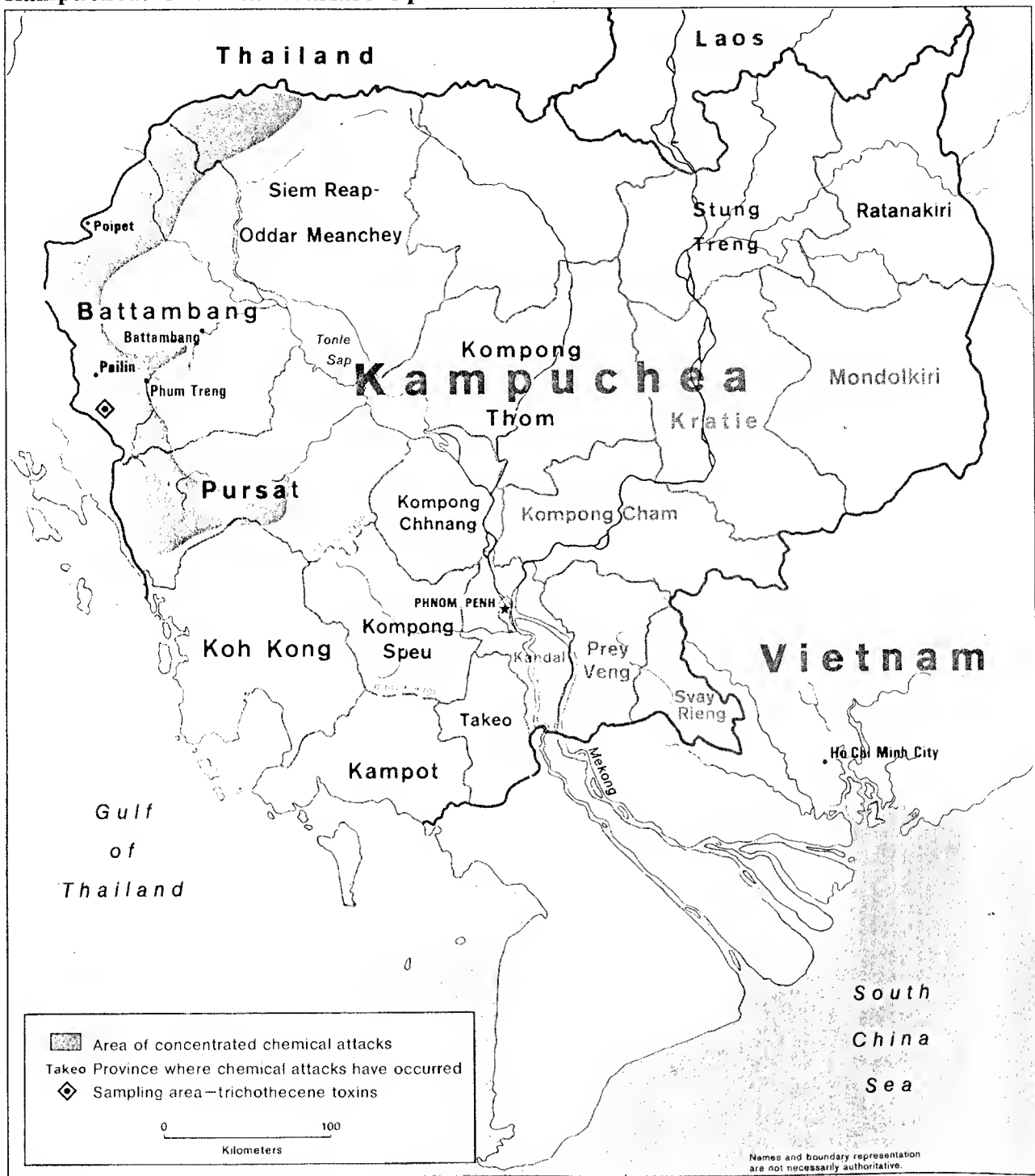
In March 1979, during Vietnamese operations against Khmer Rouge forces in the Phnom Melai area, a Vietnamese

Army private, who later defected, observed the following activities related to chemical warfare. During the fighting, all regiment (740th) troops were issued gas masks. However, the 2nd Battalion, a "border defense unit," was not issued masks. This unit was in the Phnom Melai area and was virtually surrounded by Khmer Rouge forces. At another point in the battle, the regiment's troops were ordered to don masks. The Vietnamese Army private reported that he saw two Soviets (Caucasians) fire a DH-10 (a hand-held weapon identified by the private's comrades). He was about 50 meters from the firing point. The weapon at impact, which he was able to observe from his position, gave off clouds of white, gray, and green gas/smoke. His signal unit subsequently passed a message reporting that there were 300 dead, including the unprotected Khmer Rouge and Vietnamese of the border defense forces' 2nd Battalion. The corpses reportedly had traces of white and green powder on their faces and clothes. Their faces were contorted, with eyes wide open. No blood was seen. (A H'Mong resistance leader described an incident in 1981 in which two Soviet soldiers fired a hand-held weapon that dispersed a similar lethal agent.)

Starting in February 1980, reports revealed that the Vietnamese were using 60 mm mortars, 120 mm shells, 107 mm rockets, M-79 grenade launchers filled with chemical agents, as well as munitions delivered by T-28 aircraft. According to the DK, the chemicals used were green and yellow and powderlike in appearance. In some instances the gas was described as yellow or white. The symptoms described were tightening of the chest, disorientation, vomiting, bleeding from the nose and gums, discoloration of the body, and "stiffening" of the teeth. In July 1980, the DK described artillery attacks that produced a black smoke causing itchy skin, weakness, skin lesions, and in some cases decaying skin and blisters. In December 1980, the Vietnamese were once again firing chemical artillery shells, and it was believed that poison chemicals were being brought into Thailand's border region. By March 1981, the Democratic Kampuchea forces had reported numerous attacks directed against them with lethal chemical agents and the poisoning of food and water.

U.S. analysis of contaminated vegetation samples collected within hours of a March 1981 attack showed high levels of three trichothecene toxins in a combination that would not be expected to be found in a natural outbreak in this

# Kampuchea: Chemical Warfare Operational Areas



environment. At the levels found on the vegetation, the three trichothecenes would produce vomiting, skin irritations and itching, and bleeding symptoms. Water samples taken from the area of the same attack also contained trichothecene toxins. Control samples from nearby areas confirmed that these toxins were not indigenous to the locale. (Details on the sample analysis appear in Annex D.)

There also is ample evidence of military activity at the place and time of the acquisition of the samples. Vietnamese Army defectors described plans for multiregimental sweep operations to be conducted along the border in northwestern Battambang Province before the end of the dry season in May. Actual fighting, however, continued to be characterized by guerrilla tactics on both sides, including, according to a Vietnamese Army defector, "staging ambushes, laying minefields, and use of deception." Indeed, Democratic Kampuchean resistance forces were ordered to avoid large-scale operations and to limit combat operations to scattered sapper attacks. Such information is consistent with other reports of Vietnamese Army forces spreading toxic chemicals in streams, along roadsides, and around villages and firing toxic gas shells against enemy positions. The Phnom Melai sector, where Phnom Mak Hoeun is located, was described as an "anthill of DK activity," and actions reported during March were "sporadic firefights" around Phnom Mak Hoeun involving the Vietnamese Army's 2nd Battalion, 2nd Border Security Regiment.

In Kampuchea, as in Laos, the period of late 1980 through spring 1981 was one of intensified Vietnamese operations to suppress the resistance and break the will of the opposing forces. In July 1981, trucks loaded with blue sacks filled with white powder were being moved by the Vietnamese into the Pailin, Battambang, and Siem Reap areas. Vietnamese soldiers told villagers that the chemicals caused blindness, hemorrhaging, and vomiting.

Additional evidence was derived from blood samples drawn from victims of Vietnamese chemical use that occurred on September 19, 1981 in the Takong area. Takong is in the same general area as Phnom Mak Hoeun—that is, the central region of Battambang Province near the Thai border. Although there is no independent confirmation of the accounts of the attack, American medical personnel visiting a DK field hospital examined the victims and obtained the blood samples. Analyses of these samples suggested the

use of trichothecenes. (Blood analysis results also appear in Annex D.)

According to the DK soldiers affected, the chemicals used in the September 19 Takong attack were dispersed as a gas or powder and as a poison to water. The gas or powder was released from containers by tripwires in the area of the rear forces. This description is consistent with the other reporting for this area and time.

Thailand also has been concerned about chemical attacks against its own forces and civilian population. In March 1981, one Thai died from poisons placed by Vietnamese troops, and others became ill after suffering bleeding from the nose and mouth. In May 1981, Thai forces captured two Vietnamese as they were attempting to poison the water supply in a Kampuchean relocation camp in Thailand. The poison was analyzed by the Thai and found to contain lethal quantities of cyanide. Many reports indicate that it is common practice for Vietnamese units to poison water and food used by the DK forces.

#### **The Soviet Connection in Southeast Asia**

Much of the Soviet interest in Southeast Asia is dictated by their rivalry with China and their close alliance with the Vietnamese. Regional Communist forces have been strengthened to contain Chinese influence and deter military incursions. The area of northern Laos between Vientiane and the Chinese border—where the H'Mong hill tribes have stubbornly resisted and harassed Vietnamese forces—is strategically significant to the Vietnamese because it adjoins a hostile China. In the last few years the Vietnamese have expanded their military construction and strengthened their forces in Laos which now number 50,000.

Initially there was a tendency to interpret the Soviet role as strictly advisory. Now, however, there is considerable evidence to suggest that the Soviets are far more involved in the Lao and Vietnamese chemical warfare program than was assumed earlier. An estimated 500 Soviet military advisers provide maintenance assistance and technical support, actually running the Lao Air Force, and give advanced training to Lao personnel in conventional as well as chemical warfare.

The Soviets have had advisers and technicians working in Vietnam and Laos for many years and in Kampuchea since 1979. However, it was not until

early 1979 that evidence surfaced on the Soviets' direct involvement in chemical warfare activities. For example, the Lao Army chemical section in Xiangkhoang prepared Soviet-manufactured chemical items for inspection by a Soviet military team on February 7, 1979. A seven-man team of Soviet chemical artillery experts, accompanied by Lao chemical officers, inspected chemical supplies and artillery rounds at the Xeno storage facility in Savannakhet on June 1, 1979. One report stated that the Soviets would be inspecting the same chemical explosives used to suppress the H'Mong in the Phou Bia area.

In addition to this information, H'Mong accounts have described Soviet advisers and technicians participating in the preparation of the chemical weapons for the attacks on the H'Mong villages. H'Mong eyewitnesses claim to have seen "Caucasian pilots" in aircraft, and one H'Mong report states that a downed Soviet aircraft was discovered in the jungle along with a dead Soviet pilot. In November 1981, a H'Mong resistance leader described how Soviet soldiers fighting with the Lao Army fired handheld weapons that dispersed a lethal agent over a 300-meter area. Several Lao defectors have reported seeing Soviet advisers present when aircraft were loaded with chemical-agent rockets.

In July 1981, a Soviet shipment of wooden crates filled with canisters described by the Vietnamese as "deadly toxic chemicals" was unloaded at the port of Ho Chi Minh City. This incident further corroborates the judgment that the Soviets have been shipping chemical warfare materiel to Vietnam for some time. During the unloading, Vietnamese soldiers were caught pilfering the wooden crates containing the canisters. The soldiers dropped one of the wooden cases and intentionally broke it open; they wanted to determine if its contents were edible or valuable for pilferage. When a soldier broke the nylon seal and attempted to pry open a canister, special security personnel isolated the area and told the soldiers that the canisters contained deadly toxic substances from the U.S.S.R. The wooden crates, each weighing 100 kilograms, were loaded on military trucks and taken under special guard to the Long Binh storage depot.

This incident is only one in a series involving Soviet chemical warfare materiel dating back several years. In 1975, for example, a Soviet captain of a diving support craft engaged in salvaging a sunken ship in the Black Sea, which had been transporting Soviet military supplies to Vietnam, said that

his divers came in contact with toxic chemicals, and a special Soviet salvage unit took over the operation after the divers became very ill. The salvage operations, conducted by the ASPTR-12 Salvage, Rescue, and Underwater Technical Services Group based in Odessa, were monitored by high-ranking Soviet naval officers.

The operation began with the removal of tractors and helicopters which cluttered the deck of the ship and prevented access to hold hatches. Once the surface clutter was removed, the divers attempted to enter the holds. At this point, however, operations had to be suspended temporarily because of a violent outbreak of chemical poisoning among the divers. Contact with the unidentified chemicals resulted in reddish welts 1-3 centimeters in diameter on exposed skin and was accompanied by severe headaches, nausea, and a general feeling of fatigue. The symptoms disappeared on their own after 3-5 days of rest. At this point, military authorities took over from the ASPTR-12 divers, who were temporarily withdrawn from the project. Soviet naval divers were sent down and determined that the source of poisoning was chemical seepage from an open hatch of one of the holds. The hatch was promptly sealed, and the salvage operation was once more assigned to ASPTR-12 divers who resumed work and retrieved ammunition and an assortment of other equipment. Once this was done, the military took over permanently. The ship was raised without removing the poisonous chemicals and towed to an Odessa shipyard where the chemicals were unloaded by military personnel. The ship was then broken up and scrapped. The entire operation took about 3 years to complete.

As another example of Soviet involvement, two Vietnamese corporals, from the 337th and 347th Vietnamese Army divisions, have stated that Soviet-supplied chemical weapons were stored in caves near Lang Son in February 1979. Although their Vietnamese units were issued gas masks, they were told that Soviet-supplied chemical weapons would not be used unless the Chinese initiated chemical warfare. As late as February 1981, a team of uniformed Soviet military advisers was attached to the corps headquarters. The team leader was a senior Soviet colonel. The Soviets were involved in training corps personnel in the use of Soviet-supplied weapons and equipment, including chemical artillery shells and gas masks. The Soviet team often inspected defensive positions and observed training maneuvers.

## Afghanistan

Attacks with chemical weapons against the *mujahidin* guerrillas in Afghanistan were reported as early as 6 months before the Soviet invasion on December 27, 1979. The information specifies that Soviet-made aircraft were used to drop chemical bombs, with no clear identification of Soviet or Afghan pilots or of the specific agents used. On

November 16, 1979, chemical bombs reportedly were dropped along with conventional air munitions on targets in Farah, Herat, and Badghisat Provinces by Soviet-supplied Afghan IL-28 bombers based at Shindand. A number of Afghan military defectors have stated that the Soviets provided the Afghan military with chemical warfare training

TABLE 3

### Afghanistan: Summary of Reported Chemical Attacks and Associated Deaths, 1979-81

| Time Period    | Province   | Attacks <sup>a</sup> | Deaths <sup>b</sup> |
|----------------|------------|----------------------|---------------------|
| Summer 1979    | Badakhshan | 1                    | 2,000 <sup>c</sup>  |
|                | Parvan     | 1                    | 8                   |
|                | Bamian     | 1                    | —                   |
| Fall 1979      | Konarkha   | 1                    | 350                 |
|                | Farah      | 1                    | ?                   |
|                | Herat      | 1                    | ?                   |
|                | Badghisat  | 1                    | ?                   |
| Winter 1979-80 | Badakhshan | 5                    | 130 +               |
|                | Takhar     | 1                    | —                   |
|                | Konarkha   | 2                    | 10 +                |
|                | Nangarhar  | 1                    | ?                   |
|                | Bamian     | 1                    | ?                   |
| Spring 1980    | Badakhshan | 1                    | 1 +                 |
|                | Konarkha   | 2                    | ?                   |
|                | Oruzgan    | 1                    | —                   |
|                | Qandahar   | 1                    | —                   |
| Summer 1980    | Nangarhar  | 2                    | 1                   |
|                | Vardak     | 1                    | 3                   |
|                | Herat      | 2                    | 300 +               |
|                | Kabul      | 2                    | —                   |
| Fall 1980      | Konarkha   | 1                    | ?                   |
|                | Lowgar     | 1                    | 4                   |
|                | Ghazni     | 1                    | 100                 |
| Winter 1980-81 | Lowgar     | 2                    | ?                   |
| Spring 1981    | Parvan     | 2                    | —                   |
|                | Lowgar     | 3                    | —                   |
|                | Ghazni     | 2                    | ?                   |
|                | Qandahar   | 1                    | —                   |
|                | Herat      | 1                    | —                   |
| Summer 1981    | Nangarhar  | 2                    | ?                   |
|                | Qandahar   | 2                    | 16                  |
|                | Herat      | 1                    | 119                 |
|                |            | 47                   | 3,042               |

<sup>a</sup> This tabulation omits some attacks described in the text because they could not be dated or located with high confidence.

<sup>b</sup> A plus sign indicates that the report(s) of deaths gave a minimum figure. In some cases (shown with a question mark) deaths were reported, but no number was given. Other reports (signified with a dash) gave no information on fatalities.

<sup>c</sup> The quality of reporting for this period is not as good as the information that became available after the Soviet invasion. We are concerned that this unusually high figure may reflect an accumulation of deaths from several incidents and not the single attack indicated. For example, reports were received describing over 1,000 deaths in Bamian Province in June-July 1979. An Afghan military officer reported seeing the bodies of many *mujahidin* in Panjsher Valley in August 1979 after a chemical attack and stated that many had been killed. An Afghan civil engineer reported hearing that many deaths resulted from a chemical attack in the Jalalabad area, also in the summer of 1979. Because we could not obtain supporting evidence, these reports were not included. Although sufficient evidence exists to conclude that Afghan Government forces used chemical weapons, mainly bombs, from June to December 1979, no survivors or eyewitness accounts of these attacks are available to determine the type of agent and symptoms.



as well as supplies of lethal and incapacitating agents.

For the period from the summer of 1979 to the summer of 1981, the U.S. Government received reports of 47 separate chemical attacks with a claimed death toll of more than 3,000 (see Table 3). Of the 47 reports, 36 came from Afghan Army deserters, *mujahidin* resistance fighters, journalists, U.S. physicians, and others. For 24 of the reported attacks, there is additional independent evidence supporting allegations of chemical attacks. In seven instances, further individual reporting exists. Evidence for 20 of the reported incidents comes from information on Soviet or Afghan Army combat operations in progress in areas and at times approximating those of a reported chemical attack (see map).

The reports indicated that fixed-wing aircraft and helicopters usually were employed to disseminate chemical warfare agents by rockets, bombs, and sprays. Chemical-filled landmines were

also reportedly used by the Soviets. The chemical clouds were usually gray or blue-black, yellow, or a combination of the colors.

Symptoms reported by victims and witnesses of attacks indicate that non-lethal incapacitating chemicals and lethal chemicals—including nerve agents, phosgene or phosgene oxime, possibly trichothecene toxins, and mustard—were used. Medical examinations of some of the victims include reports of paralysis, other neurological effects, blisters, bleeding, and sometimes death. While none of the agents being used in Afghanistan has been positively identified through sample analysis, there is no doubt that the agents being used are far more toxic than riot-control agents such as CN and CS or even adamsite.

Several descriptions of the physiological action of a chemical agent or of the condition of the corpses of victims were particularly unusual. In one, victims were rapidly rendered unconscious for 2-6 hours and had few

aftereffects. In another, the bodies were characterized by abnormal bloating and blackened skin with a dark-reddish tinge, and the flesh appeared decayed very soon after death. In a third incident, three dead *mujahidin* guerrillas were found with hands on rifles and lying in a firing position, indicating that the attacker had used an extremely rapid-acting lethal chemical that is not detectable by normal senses and that causes no outward physiological responses before death.

Shortly after the Soviet invasion, many reports were received that both Soviet and Afghan forces were using various types of chemical agents. Ten separate chemical attacks, resulting in many deaths, were reported in the first 3 months of 1980. These reports came from northeastern Afghanistan and provide the highest percentage of reported deaths. During the mid-January to February 1980 period, helicopter attacks were reported in northeastern Afghanistan in which a grayish-blue smoke resulted in symptoms similar to those

described by the H'Mong refugees from Laos (e.g., heavy tearing or watering of eyes; extensive blistering and discoloration of the skin, later resulting in large sheetlike peeling; swelling in the areas affected by the blister; and finally numbness, paralysis, and death). Medical reports from examinations in Pakistan of refugees from a large attack in the upper Konar Valley in February 1980 described red skin and blisters containing fluid described as "dirty water." Refugees estimated that about 2,000 people were affected after contact with a dirty yellow cloud.

By spring and summer of 1980, chemical attacks were reported in all areas of concentrated resistance activity. Many reports from different sources strongly support the case that irritants were used to drive the insurgents into the open to expose them to attack with conventional weapons and incapacitants to render them tractable for disarming and capture. On several occasions in April 1980, for example, Soviet helicopter pilots dropped "gas bombs" on insurgents, evidently to drive them from caves.

A Dutch journalist, Bernd de Bruin, published an eyewitness account of two chemical attacks occurring in the Jalalabad area on June 15 and June 21, 1980 (*Newsnet*, August 2, 1980). He filmed an MI-24 helicopter dropping canisters that produced a dirty yellow cloud. A victim with blackened skin, discolored by extensive subcutaneous hemorrhaging, was photographed in the village 5 hours after the attack. The journalist evidently was exposed because he developed blisters on his hands and a swollen and itchy face. He also was exposed in the second attack, and it took about 10 days for him to recover from skin lesions, nausea, diarrhea, and stomach cramps.

An Afghan insurgent provided an eyewitness account of a July 6, 1980 attack on a village 10 kilometers east of Darae Jelga in Vardak Province. He reported that a Soviet MI-24 helicopter gunship dropped a bomb that, upon explosion, released a lethal chemical. A separate report confirmed that Soviet bombing attacks on villages in Vardak as well as Lowgar and Parvan Provinces were taking place during this period. In August 1980, information surfaced on a Soviet attack with chemical bombs on the village of Sya Wusan, 30 kilometers southeast of Herat, leaving 300 dead. It was during this time that the Soviet chemical battalion at Shindand set up an operational decontamination station.

Reports of chemical weapons use in 1981 essentially parallel 1980 reporting with respect to frequency and location of

attack. Soviet helicopter units participated in chemical attacks from April 20 to April 29, 1981, in areas east and west of Kabul and in the Konar Valley, according to eyewitness accounts. These attacks were intended to drive personnel from sanctuaries, such as caves, in order to engage them with conventional fire. The munitions were described as Soviet 250-kilogram RBK cluster bombs. The Soviets have such a munition, which can be filled with chemical agents. Other reports described similar operations by helicopters north of Qandahar on April 24 and April 26, 1981.

A former Afghan MI-8 helicopter pilot said Soviet forces had used chemical weapons in Badakhshan, Qonduz, and Konarha. Chemicals in canisters that contained toxic gas, tear gas, and antirespiratory gas, which has an incapacitating effect by causing choking and difficulty in breathing, were manually pushed from the cargo compartment of helicopters. The pilot said that there also was a specific gas that is absorbed by the body and leaves the skin so soft that a finger can be punched through it. In one case, there was a wind shift, and Soviet and Afghan forces were seriously affected. Other sources also have described an incident where Soviet and Afghan forces were victims of their own gas attack.

The following sequence occurred in a small valley in Qandahar Province in early June 1981. According to an Afghan exile, Soviet combat groups engaged rebel forces in that valley during a 2-week period. The situation worsened for the Soviets, and an airstrike was conducted. The exile stated that a Soviet helicopter delivered a single rocket, releasing a chemical that killed 16 insurgents. Nearly all reports state that chemicals were delivered by aircraft or helicopters; a few reports describe chemical artillery rounds.

Before a sweep operation in the Konar Valley in September 1981, resistance leaders were told by an Afghan officer that the Soviets had four agents available but would use only the incapacitant which they could defend against with wet rags over the face. During the operation, Soviet helicopters conducted gas attacks in 25 different areas, using cylinders about 1.5 meters long and 60 centimeters in diameter that exploded 4-5 meters above the ground, releasing the incapacitating gas. Some victims lost consciousness, were paralyzed, and recovered, but others died, and unprotected areas of their skin turned dark green to blue-green.

An Afghan tribal leader recently described a Soviet chemical attack against a large resistance force in October 1981

near Maruf, about 100 kilometers east of Qandahar. Soviet helicopters dropped green cylindrical canisters (18 inches long, 3-4 inches in diameter) which, upon hitting the ground, emitted a greenish-yellow gas. According to the report, victims felt faint and dizzy; later their skin began to itch, and many lost consciousness. About 300 persons were affected by the gas and many died. Soviet ground forces captured many of the survivors. Other information on Soviet and *mujahidin* activities in the Qandahar area during this period confirms that this incident did in fact take place.

In February 1982, a member of the resistance, with considerable knowledge of Soviet weapons, told a U.S. official that the Soviets were using irritants, a hallucinogenic gas, and what he said was an apparent nerve gas. He described the "nerve agent" as an off-white powdery substance dispersed from helicopters generally during artillery or bombing attacks. Victims realize they have been exposed to chemical attack only when they become faint and dizzy. Subsequently, they begin to vomit and bleed from the eyes, nose, and mouth. Death occurs within a short time. The corpses are extremely relaxed, with no evidence of rigor mortis. Flesh and skin frequently peel off if an effort is made to move the bodies.

According to this account, survivors suffer aftereffects for about 6 months, including chest congestion and pain, dizziness, and mental agitation. The powder-like substance is more effective at lower altitudes where there is less wind to dilute the poison, and *mujahidin* groups have experienced fatality rates as high as 70%. Many survivors of chemical attacks in Laos and Afghanistan have exhibited the same long-term health problems described in this account.

Chemical defense battalions—standard in all Soviet divisions—are deployed with the three Soviet motorized rifle divisions operating in Afghanistan at Qonduz, Shindand, and Kabul. Soviet operational personnel decontamination stations were observed at several locations, and chemical decontamination field units were deployed during a sweep operation of the Konar Valley in eastern Afghanistan and near Shindand in the west in 1980. The operational deployment of decontamination units for personnel and equipment suggests that chemical battalions have supported offensive chemical use. In addition, Soviet personnel have been observed wearing chemical protective equipment. The Soviets have specifically tailored their forces in Afghanistan, in part

because of logistical constraints; 5,000 troops and "nonessential" combat equipment were withdrawn, but the chemical battalions remain.

A Soviet military chemical specialist, captured by the *mujahidin*, gave his name as Yuriy Povarnitsyn from Sverdlovsk. During an interview, he said that his mission was to examine villages after a chemical attack to determine whether they were safe to enter or required decontamination. An Afghan pathologist who later defected described accompanying Soviet chemical warfare personnel into contaminated areas to collect soil, vegetation, and water samples after Soviet chemical attacks. According to firsthand experience of former Soviet chemical personnel, the Soviets do not require decontamination equipment in an area where chemical bombs are stored or loaded onto aircraft. Thus, deployment of this equipment in Afghanistan must be assumed to be associated with the active employment of casualty-producing chemical agents.

Afghan military defectors have provided information on ammunition and grenades containing phosgene, diphosgene, sarin, and soman and have described where and when some of them have been used. They also have revealed locations where these agents were stockpiled. The agents used, plus the time and location of the attacks, correspond with the refugee reports and recorded military operations.

The Soviet Union has stocked a variety of toxic chemical agents and munitions to meet wartime contingencies. Weapons systems capable of delivering chemical munitions available to Soviet forces in Afghanistan include artillery, multiple rocket launchers, and tactical aircraft.

### Motivation for Using Chemical Weapons

In the course of this analysis, the question has been posed: Is there a military-strategic or tactical rationale for the systematic use of chemical weapons by conventional forces in Laos, Kampuchea, and Afghanistan? The military problems faced in these countries—viewed from the perspective of the Soviets and their allies—make the use of chemical weapons a militarily effective way of breaking the will and resistance of stubborn anti-government forces operating from relatively inaccessible, protected sanctuaries.

The Soviets have made a large investment in insuring that Vietnam and its clients succeed in extending their control over Indochina. For Vietnam, the H'Mong resistance in Laos is a ma-

jor irritant to be removed as quickly and cheaply as possible. The use of chemical agents has played a major role in driving the H'Mong from their mountain strongholds, relieving Vietnamese and Lao ground forces of the need for costly combat in difficult terrain. Much of the H'Mong population that lived in the Phou Bia mountain region has been driven into Thailand, killed, or resettled.

In the mountainous areas of Afghanistan, where rebels are holed up in caves or other inaccessible areas, conventional artillery, high-explosive bombs, and napalm are not particularly effective. Many reports indicate that unidentified chemical agents have been used on such targets. Caves and rugged terrain in Laos and thick jungles in Kampuchea also have frustrated attempts to locate and destroy the resistance forces. Chemical clouds can penetrate the heavy forests and jungle canopy and seep into the mountain caves. Persistent agents linger in the area and cause casualties days and sometimes weeks after the attack. Unprotected forces and civilians have little or no defense against lethal agents like toxins, nerve gas, or blister agents.

Trichothecene toxins, which are known to have been used in Southeast Asia, have the added advantage of being an effective terror weapon that causes bizarre and horrifying symptoms. Severe bleeding, in addition to blisters and vomiting, has instilled fear in the resistance villages. Not only have the villagers and their animals been killed in a gruesome manner, but the vegetation and water also have been contaminated. Survivors are reluctant to return to their inhospitable homes and instead make the long and dangerous trek to camps in Thailand.

There is no clearcut explanation of why trichothecene toxins have been used in addition to irritants, incapacitants, and other traditional chemical warfare agents. Speculation suggests that they are probably cheaper to make and are readily available from Soviet stocks; they are probably safer and more stable to store, transport, and handle in a Southeast Asian environment, and they may require less protective equipment when being prepared for munitions. They are difficult to trace as the causative agent after an attack—as demonstrated by the length of time it took for the United States to detect them. Few laboratories in the world have the analytical capability to identify precisely the type and amount of trichothecene toxin in a sample of vegetation, soil, or water.

The Soviets may well have calculated that they and their allies

could successfully deny or counter charges that chemical weapons had been used, recognizing that it would be especially difficult to compile incontrovertible evidence from inaccessible areas of Southeast Asia and Afghanistan. With respect to Kampuchea, they may also have calculated that, in view of the lack of international support for Pol Pot's resistance, chemical weapons could be used on his troops without significant international outcry.

In addition, the Soviet military very likely considers these remote areas as providing unique opportunities for the operational testing and evaluation of chemical weapons under various tactical conditions. Years of aerial and artillery chemical dispersion have undoubtedly provided the Soviets with valuable testing data. Southeast Asia has offered the Soviets an opportunity to test old agents that had been stockpiled for many years as well as more recently developed agents or combinations of agents. This conclusion is supported by information from foreign military officers who have attended the Soviet Military Academy of Chemical Defense in Moscow. According to their Soviet instructor, three types of chemical agents may be used during the "initial stages" of local wars: "harassing agents (CS, CN, DM), incapacitants such as psychochemicals (BZ) or intertoxins [sic—possibly enterotoxins], and herbicides." During the "decisive phase, lethal agents can be employed under certain circumstances." In a local war, "chemical weapons can be used to spoil enemy efforts to initiate operations, even if the enemy has not used them first." The foreign officers' accounts, including detailed descriptions of the Soviet chemical warfare program, support the conclusion that the Soviets consider chemical weapons an effective and acceptable means of warfare in local conflicts.

Insight into the Soviet bloc military perspective on the use of toxins is provided in the following passage from a 1977 East German military manual entitled *Textbook of Military Chemistry*.

Toxins are designated as toxic agents which are produced by biological organisms such as micro-organisms, plants, and animals, and cannot themselves reproduce.

By the middle of 1960 the toxins selected for military purposes were included among the biologic warfare agents. In principle, this was understood to mean only the bacterial toxins. Today it is possible to produce various toxins synthetically. Toxins with 10-12 amino acids can currently be synthesized in the laboratory. Toxins are not living substances and in this sense are chemicals. They thus differ fundamentally from the biological organisms so that they can be included among chemical warfare agents. As a result

of their peculiarities they are designated simply as "toxin warfare agents." They would be used in combat according to the same principles and with the same methods used for chemical warfare agents. When they are used in combat the atmosphere can be contaminated over relatively large areas—we can expect expansion depths of up to 6 kilometers before the toxin concentration drops below lethal concentration 50 . . . the toxin warfare agents can be aerosolized. They can be used primarily in micro-bombs which are launched from the air or in warheads of tactical rockets. Toxin warfare agents concentrates can be applied with aircraft spray equipment and similar dispersion systems.

The Soviet designation for several pathogenic *Fusarium* products is "IIF" (*iskusstvennyy infektsionnyy fon*), which stands for "artificial infection background." IIF devices are used in the Soviet Union deliberately to contaminate soil in experimental agricultural test areas with spores of disease-producing fungi. We are not certain if the IIF compounds include trichothecenes. Nor are we certain as to the intent of this agricultural research program. It is possible that these programs are designed to colonize soil with pathogenic organisms either to determine which crop varieties are most resistant to disease or, alternatively, to test eradication and control methods in infected soils. Elsewhere in the Soviet agricultural research program, however, it is known that there is widespread use of certain trichothecenes, including sprays from light aircraft. A capability exists within the Soviet Union for multi-ton production of light aircraft spray-delivered microbial products such as those described above.

Evidence accumulated since World War II clearly shows that the Soviets have been extensively involved in preparations for large-scale offensive and defensive chemical warfare. Chemical warfare agents and delivery systems developed by the Soviets have been identified, along with production and storage areas within the U.S.S.R. and continuing research, development, and testing activities at the major Soviet chemical proving grounds. Soviet military forces are extensively equipped and trained for operations in a chemically contaminated environment. None of the evidence indicates any abatement in this program. The Soviets have shown a strong interest in improving or enhancing their standard agents for greater reliability and effect. Their large chemical and biological research and development effort has led them to investigate other kinds of chemical warfare agents, particularly the toxins.

None of the four countries considered in this report—Vietnam, Laos,

Kampuchea, and Afghanistan—has any known large-scale facility or organization for the manufacture of chemical and biological materials. Nor are they known to have produced even small quantities of chemical warfare agents or munitions. The technical problems of producing large quantities of weapons-grade toxins, however, are not so great as to preclude any of the four countries from learning to manufacture, purify, and weaponize these materials. It is highly unlikely, however, that they could master these functions without acquiring outside technical know-how.

## ANNEX A

### A LAO PILOT'S ACCOUNT

One of the most complete descriptions of chemical warfare activities in the 1976-78 period came from a Lao pilot who was directly involved in chemical warfare. The pilot, a former Lao People's Liberation Army (LPLA) officer who defected in 1979, reported that he flew captured L-19 and T-41 aircraft equipped to dispense toxic chemical agents on H'Mong villagers in the Phou Bia area of northern Laos. He said that the LPLA, in cooperation with the Vietnamese Army, had conducted chemical warfare operations in Laos since April or early May 1976. At that time, two Lao H-34 helicopters were flown between Long Tieng and the Phonsavan airfield, both in Xiangkhoang Province, on a series of flights to transport rockets to Phonsavan for storage.

Between June and August 1976, the LPLA launched attacks in the area of Bouamlong—in Xiangkhoang Province—a stronghold for remnants of the forces of former H'Mong Gen. Vang Pao. The LPLA used L-19 aircraft for rocket attacks in that area aimed at eliminating the H'Mong resisting government control. Lao crews responsible for loading rockets on the attack aircraft noted that they were not allowed to use the rockets that had been moved from Long Tieng to Phonsavan, even though Phonsavan was much closer to the Bouamlong target area than Long Tieng, where Lao aircraft had to rearm. The pilot said that, during nearly 3 months of flying missions against the Bouamlong area, he flew his L-19 aircraft to Long Tieng to be armed with rockets.

In late 1976, the pilot's L-19 aircraft was armed with rockets stored at Phonsavan. Initially, H-34 helicopters were used to transport the rockets from Phonsavan to a depot near the Ban Xon

airfield (Vientiane Province), where the rockets were fitted onto racks of the L-19 aircraft for missions in the Phou Bia area; later, the rockets from Phonsavan were transported to Ban Xon by trucks. All U.S.-manufactured rockets were stored with the tip and canister kept apart; the two parts had to be joined before being fitted to the racks on the aircraft. The pilot observed, however, that all the rockets transported from Phonsavan to Ban Xon were already assembled.

As part of his routine flight activities, the pilot would check his aircraft and, in doing so, examine the tip portion of new smoke rockets that had been transported from Phonsavan. He said that most appeared "loose" in the portion where the tip and canister joined, whereas the tip and canister of the ordinary explosive-type rockets at Long Tieng were noticeably more tightly connected.

In late 1976, during preparation for airstrikes on Kasy (Louangphrabang Province) and in new areas of Phou Bia, the pilot said he began carrying two or three Vietnamese Army staff officers, sometimes accompanied by a Lao staff officer, in T-41 aircraft for reconnaissance over the target areas. When these airstrikes were launched, the defector pilot initially flew his L-19 aircraft on missions with another pilot and a Lao staff officer. After 2 or 3 weeks, however, Vietnamese staff officers, who spoke excellent Lao, began alternating with the Lao officers. Before each mission, the Vietnamese or Lao staff officer would go over target areas outlined on situation maps—which then were taken along—and would point out the targets to be attacked. The defector pilot noted that at no time did the Vietnamese staff officer communicate with Lao officers on the ground, as did the Lao staff officers. A new Vietnamese officer was assigned for each airstrike mission in the H'Mong areas.

The pilot related that before flying L-19 airstrike missions with a full load of rockets he was often warned by a Lao commander to fly at above-normal altitudes when firing rockets—to preclude hazard to the occupants of the aircraft. For this reason the pilot surmised that the "smoke" rockets fired at the H'Mong were unusual. He was able to observe that the "smoke" rockets detonated in the air and that some produced white smoke, with a mixture of blue, while others produced red smoke, with a mixture of yellow. The ordinary explosive-type rockets detonated on impact. The

commander or his designated representative told the pilot before every mission that the operations—called Extinct Destruction Operations—were intended to “wipe out the reactionary H'Mong people.”

Before a mission involving “smoke rockets,” the commander warned the pilots to keep the operation secret. The Lao defector said that, during the nearly 2 years in which he flew rocket missions, he learned from the Lao staff officers accompanying him that there were two types of rockets. The first, mostly “smoke” rockets, were to be fired at targets far away from Lao and Vietnamese troops to avoid exposing them to the poison smoke. The second was of the ordinary explosive type, considered a “close support” rocket that could be fired near Lao troop positions. Initially, the L-19 aircraft carried eight rockets—five “close support” and three “smoke” rockets. Later, only four rockets, mainly of the “smoke” type, were carried.

After each mission in which chemical warfare rockets were used, the pilot was returned to a “rest house” at Phonsavan, where a Lao Army doctor and nurse would examine him. He said that after his missions, especially in 1978, he was particularly well treated by the examining doctor and watched very closely by the nurse. Those L-19 aircraft pilots assigned to missions utilizing chemical warfare rockets had special privileges, including additional flight pay and free meals at the Phonsavan cafeteria. In October 1978, the Lao Army stopped using L-19 aircraft on combat missions and began using Soviet MiG-21s for chemical attacks on the Phou Bia areas.

Several H'Mong reports corroborate the testimony of the Lao pilot. A village chief, for example, described attacks covering all 7 days of the week of June 5, 1976 in the Bouamlong area. He described L-19 aircraft firing rockets that produced red and green smoke: Ten villagers were killed by gas and 30 by shrapnel. Most of the H'Mong reports documented by a U.S. Foreign Service officer in June 1979 and a Department of Defense medical team in October 1979 were consistent with the pilot's testimony. H'Mong observers familiar with military aircraft reported L-19s in use until late 1978. After that time, reports described jets or “MiGs” and some accurately described Soviet AN-2s.

A review of information back to 1975 shows L-19 and T-28 aircraft were operating from airfields in

northern Laos—including the one at Phonsavan, where AN-2s were seen in 1978. Failure to observe chemical decontamination equipment at the airfields does not rule out the presence or handling of chemical munitions. The Soviets supervise the chemical warfare activities in Laos; it is assumed that chemical munitions are handled in about the same manner as in the U.S.S.R. According to former Soviet chemical warfare personnel, no protective clothing or special decontamination equipment is required for loading chemical bombs onto aircraft and helicopters at chemical munitions test ranges.

The Lao pilot's description of the rockets used on the L-19 was corroborated by other sources. A H'Mong refugee, a former commander of a 500-man resistance force, reported that in 1977 he found a rocket canister and a separated warhead that he believed were the kinds used by the Vietnamese and Lao. The canister had authentic U.S. markings identifying it as a U.S.-manufactured 2.75-inch rocket and, reportedly, three lines of Russian writing which he could not translate. Another H'Mong resistance force officer, reportedly trained as a liaison officer and ordnance expert before the Communist takeover of Laos, stated that he, too, believed that the rocket canister was of U.S. manufacture and that the Soviet technicians in Laos had modified the upper stage to contain a poisonous (i.e., lethal) chemical.

The diameter of the warhead was reported to be 12.5 centimeters (5 inches), probably a measurement taken on a modified warhead, because the United States does not have a 5-inch warhead for the 2.75-inch “rocket motor.” During the Vietnam conflict, about 35 million U.S.-manufactured, conventional 2.75-inch rockets were sent to the war zone, and many tens of thousands of these fell into North Vietnamese hands when the South Vietnamese forces collapsed. The Vietnamese may be using some of these rockets with existing loads, but modified warheads for the 2.75-inch rocket motor could easily be fabricated in Vietnam and filled with a lethal or nonlethal agent in Laos, especially with Soviet assistance. According to U.S. experts, fabrication of a warhead 5 inches in diameter, necked down to fit the 2.75-inch rocket, could be accomplished by trained technicians in a small, well-equipped machine shop and laboratory.

## ANNEX B

### FINDINGS OF U.S. GOVERNMENT INVESTIGATIVE TEAMS: USE OF CHEMICAL AGENTS AGAINST THE H'MONG IN LAOS

#### State Department Team

In May 1979, State Department officials visited Thailand to interview H'Mong refugees and investigate allegations of the use of chemical agents against H'Mong tribesmen in Laos (see Table B-1). From the signs/symptoms described and observed, it is suggested that at least two and possibly three different chemical agents may have been used, such as:

- A nerve agent (five or six individuals reported symptoms that could be attributed to a nerve agent);
- An irritant or riot-control agent (one-third of the interviews); and
- More than half of the interviews indicated such a variety of signs and symptoms that it is difficult to attribute them to a single known agent.

It is possible that in some cases two or more agents were combined.

- Reported signs and symptoms suggesting a nerve agent include sweating, tearing, excessive salivation, difficulty in breathing, shortness of breath, nausea and vomiting, dizziness, weakness, convulsions, and death occurring shortly after exposure.
- Reported signs and symptoms suggesting a riot-control or irritant agent include marked irritation or burning of the eyes, with tearing and pain; irritation and burning of the nose and throat; coughing; burning and tightness in the chest; headache; and nausea and vomiting in a few cases.
- Reported signs and symptoms not related to any known single agent include a mixture of the above as well as profuse bleeding from mucous membranes of the nose, lungs, and gastrointestinal tract, with rapid death of the affected individuals in some instances.

Estimates from the H'Mong interviewed indicate that approximately 700-1,000 persons may have died as a result of the use of chemical agents and that many times this number became ill. It was reported that on many occasions entire villages were devastated by these agents, leaving no survivors.

In the episodes described, most of the animals exposed to the chemical agents were killed. Generally, all

chickens, dogs, and pigs died and, to a lesser extent, the cattle and buffalo. On several occasions it was reported that where these agents settled on tree and plant leaves, many small holes appeared in the leaves within 2 or 3 days. Rarely did agent exposure result in the defoliation or death of the plants.

#### Department of Defense Team

From September 28 to October 12, 1979, a team from the U.S. Army Surgeon General's Office was in Thailand to conduct a similar series of interviews.\* The team visited the following H'Mong refugee camps of northern Thailand: the detention center at Nong Kai, the large H'Mong camp at Ban Vinai, and two smaller camps at Nam Yao and Mae Charim. As the great majority of refugees as well as the H'Mong leadership are at Ban Vinai, most interviews were obtained there.

The team was prepared to obtain blood and skin samples (for cholinesterase activity and study of pathological changes, respectively) from those exposed to chemical agents. For such samples to yield meaningful results they must be taken within 6-8 weeks of exposure. Since the last reported exposure was in May 1979, no blood or skin samples were collected.

Interviews were conducted through interpreters; one was an employee of the U.S. Consulate at Udorn, and the remainder were hired from among the refugees. The interpreters screened those refugees who volunteered to talk to the team and selected only those who had been eyewitnesses to or had themselves been exposed to an agent attack. Team members interviewed 40 men, 2 women, and a 12-year-old girl. Each interview took 1-2 hours. To insure conformity, a prepared questionnaire was used as a guide.

The chemical attacks reportedly occurred between June 1976 and May 1979 (Table B-1). The absence of reports of attacks after May 1979 may be because

\* The authors of the U.S. Army Surgeon General's report are Charles W. Lewis, M.D., COL, MC, Chief, Dermatology Service, Brooke Army Medical Center, Fort Sam Houston, Texas; Frederick R. Sidell, K.D., Chief, Clinical Resources Group, U.S. Army Biomedical Laboratory, Aberdeen Proving Ground, Md.; William D. Tigertt, M.D. (Brigadier General, Ret., USA), Professor of Pathology, University of Maryland, Baltimore, Md.; Charles D. Lane, LTC, Southeast Asia Desk Officer, OACSI, Department of the Army, Washington, D.C.; and Burton L. Kelley, SP5, USA, Dermatology Technician, Brooke Army Medical Center, Fort Sam Houston, Texas.

TABLE B-1

#### Reports of Probable Chemical Agent Attacks in Laos

##### Department of State Interviews Conducted in Summer 1979

| Date               | Location                    | Method of Attack by Plane | Material Used (Smoke/Gas)   |
|--------------------|-----------------------------|---------------------------|-----------------------------|
| Oct. 1977          | Phu Hay, S. of Phou Bia     | Rockets                   | Yellow-gray                 |
| 1978               | Pa Sieng, S. of Phou Bia    | Bomb                      | Yellow                      |
| Feb. 1978          | Ban Nam Luk, S. of Phou Bia | Spray (?)                 | Yellow/white                |
| Feb. 1978          | 20 kms SE. of Phou Bia      | Spray (?)                 | Yellow                      |
| Feb. 1978          | Ban Ko Mai                  | Bomb                      | Yellow                      |
| Mar. 1978          | Pha Houei                   | Sacks, burst in air       | Brown                       |
| Mar. 1978          | Ban Na Pong                 | (?)                       | Yellow                      |
| Apr. 1978          | Ban Phamsi                  | (?)                       | White, green, blood-colored |
| May-Apr. 1978      | Ban Nong Po                 | Cloud                     | Yellow-brown like rain      |
| June 1978          | Ban Nam Teng                | Rocket (?)                | Yellow                      |
| June 1978-May 1979 | Ban Don area                | Spray                     | Yellow                      |
| Mid-1978           | 1-3 kms NE. of Phou Bia     | Rocket, air burst         | Red                         |
| Oct. 1978          | Nam Kham                    | Rockets, air burst        | Yellow                      |
| Oct. 1978          | 6 kms N. of Phou Khao       | Rockets, air burst        | Red                         |
| Oct. 1978          | 3-4 kms N. of Phou Bia      | Rockets, air burst        | Yellow-gray                 |
| Nov. 1978          | Phou Xang Noi               | Spray                     | Yellow, blue                |
| Nov. 1978          | near Phou Bia               | Bomb, air burst           | Yellow                      |
| Nov. 1978          | NE. of Pha Khao             | Rocket, air burst         | Yellow                      |
| Apr. 1979          | Ban Nouia Pong              | Spray                     | Yellow                      |
| May 1979           | Nam Po                      | Spray                     | Yellow                      |
| May 1979           | Pha Mai                     | Spray, air burst          | Yellow                      |

##### Department of Defense Interviews Conducted in Fall 1979

| Date                      | Location         | Method of Attack by Plane | Material Used (Smoke/Gas) |
|---------------------------|------------------|---------------------------|---------------------------|
| June 1976                 | Pou Mat Sao      | Rockets                   | Red, green                |
| Jan. 1977-Oct. 1978       | Pha Khao         | Rockets                   | Yellow, red, green        |
| Mar. 1977                 | Nam Theuna       | Rockets                   | Red, yellow               |
| Apr. 1977                 | Houi Kam Lang    | Rockets                   | Yellow                    |
| May 1977                  | Pha Khae         | Rockets                   | Red                       |
| May 1977                  | Nam Moh          | Rockets                   | Yellow                    |
| May 1977                  | Pha Ngune        | Spray/rockets             | Yellow                    |
| 1977-1978 (3 attacks)     | Phu Seu          | Rockets                   | Red, green, yellow        |
| Jan. 1978                 | Houi Xang        | Rockets                   | Red, green                |
| Feb. 1978                 | Sane Mak Ku      | Rockets                   | Yellow                    |
| Feb. 1978                 | Tham Se Sam Leim | Rockets                   | Yellow, black             |
| Feb. 1978                 | Kio Ma Nang      | Rockets                   | Yellow                    |
| Mar. 1978                 | Mouong Ao        | Rockets                   | White                     |
| Mar. 1978                 | Khieu Manang     | Rockets                   | Green                     |
| Apr. 1978                 | Tha Se           | Rockets                   | White                     |
| June 1978                 | Pha Phay         | Rockets                   | Yellow                    |
| June 1978                 | Phou Seng        | Rockets                   | Red, white, black         |
| July 1978                 | Phou Bia         | Rockets                   | Red                       |
| July 1978                 | Ban Nam Mo       | Spray                     | Yellow                    |
| July 1978                 | Phou Lap         | Rockets                   | Yellow                    |
| Aug. 1978                 | Pha Houai        | Rockets                   | Red, green                |
| Aug. 1978                 | Ban Thin On      | Rockets                   | Green, red                |
| Aug. 1978                 | Bouamlong        | Rockets                   | Red, green, yellow        |
| Sept. 1978                | Pha Koung        | Rockets                   | Yellow                    |
| Sept. 1978                | Ban Nam Tia      | Spray/rockets             | Yellow, green, red        |
| Sept. 1978                | Pha Na Khum      | Rockets                   | Red                       |
| Oct. 1978                 | Phou Bia         | Rockets                   |                           |
| Oct. 1978                 | Ban Done         | Spray                     | Yellow                    |
| Oct. 1978                 | Phou Bia         | Rockets                   | White, green, red         |
| Nov. 1978                 | Phou Bia         | Rockets                   | White, red                |
| Feb. 1979                 | Pha Mat          | Spray                     | Yellow                    |
| Feb. 1979                 | Tong Moei        | Rockets                   | Yellow, red               |
| Mar. 1979                 | Pha Mai          | Spray                     | Yellow                    |
| Mar.-May 1979 (6 attacks) | Pha Mai          | Spray                     | Yellow                    |
| Apr.-May 1979 (4 attacks) | Pha Mai          | Spray                     | Gray-white                |
| May 1979                  | Phou Bia         | Spray                     | Yellow                    |
| May 1979                  | Moung Phong      | Rockets                   | Red                       |

few refugees crossed the Mekong River after that time—as a result of heavy rains and flooding from June to September 1979. Most of the early reports were of the use of rockets releasing the agent; beginning in the fall of 1978, the majority of the attacks were carried out by aircraft spraying a yellowish substance which “fell like rain.” The attack sites, concentrated around the H'Mong stronghold in the mountainous Phou Bia area, also are listed in Table B-1.

The team was given a plastic vial containing pieces of bark, stained by a yellow substance, which several H'Mong refugees claimed was residue from an aircraft spray attack in April 1979. Preliminary chemical analysis of the sample indicates that no standard chemical agent (i.e., an agent listed in TH 8-285, U.S. Army, May 1974) was present.

### Conclusions

The conclusions of these teams, based upon interviews obtained from H'Mong refugees, are as follows:

- Chemical agents have been used against the H'Mong.
- The reported effects of these agents suggest the use of a nerve agent, a riot-control agent, and an unidentified combination or compound.

## ANNEX C

### MEDICAL EVIDENCE

#### Southeast Asia

Since 1975, many different sources—refugees, relief workers and medical personnel, including specially qualified physicians—consistently have detailed unusual signs and symptoms of victims of “yellow rain.” Specifically, victims in Southeast Asia subjected to a direct attack of the yellow powder, mist, smoke, or dust would be seen to begin retching and vomiting within minutes. These effects and those described below were not pronounced in individuals even 100 meters from the attack zone, indicating a relatively dense chemical/carrier combination that was effective in low wind conditions.

Following the victim's exposure to yellow rain, the initial induced vomiting—unlike that caused by a traditional riot-control nausea agent—was protracted over hours to days. It was often accompanied by dizziness, rapid heart-beat and apparently low blood pressure, chest pain, loss of far-field vision, and a feeling of intense heat and burning on the skin, although not described as being

most acute in the groin and axillae. Thus, the acute signs and symptoms match some effects of traditional vomiting and blister agents but clearly not all.

Within the first hours after the attack, many victims also reported intense red eyes, bleeding gums, convulsions or more often trembling, and vomiting of blood, with or without production of copious amounts of saliva—lasting many hours to days, apparently depending on the exposure level. Thick mucous, pinpoint pupils, respiratory collapse, prolonged spasticity, and involuntary urination or defecation were never reported after a yellow rain attack; the absence of these symptoms helped to rule out organophosphate nerve agents in the minds of chemical warfare experts. Many medical and environmental samples also ruled out these and other traditional agents such as DM, DS, and others.

Many observers of “yellow rain” effects reported formation within several hours of small (1 centimeter) homogeneous, hard, fluid-filled blisters over only exposed areas of skin, frequently including the victim's hands, arms, entire throat, and face—wherever skin was uncovered. In most cases the vomit, after 2-8 hours, contained blood and, in many cases, large amounts of it. About half of those receiving the most concentrated doses of yellow material—those who had been directly under the spray—were observed within several hours to cease vomiting temporarily. This interval was often followed in 5-15 minutes by a period of great pain when the victim would hold his abdomen and emit a gush of blood from mouth and nose. These individuals usually died within minutes afterward.

Close questioning by physicians of witnesses to these final moments leaves no doubt that the effects resulted from severe gastrointestinal bleeding, significant pulmonary bleeding, temporary compression of accumulated blood in the stomach, and, finally, projectile vomiting of as many as several hundred milliliters of blood. These findings were consistent with animal and human autopsies.

Many victims of the yellow material received less than the full brunt of a spray, entered the attack zone several hours to 2 days later, or consumed food or water contaminated by the material. These individuals—often within the next 24 hours—developed signs and symptoms similar to those more directly affected but often without pronounced skin effects if they had not contacted the powder residue directly. In addition to

attacks of intense vomiting five or six times a day, they also had diarrhea, with bloody stools passed up to eight times a day. Bleeding under the fingernails and around the skin of the eyes and severe bruising of the skin also were commonly reported. Opiates helped the fluid loss in adults, but in children or young persons unable to tolerate the treatments of raw opium and water, death occurred after 10 days to 2 weeks in about half the cases. On the basis of reported signs and symptoms, the cause of delayed death almost certainly was dehydration.

In many cases, chemical attacks are reported to produce symptoms other than those described here. However, there has always been a direct association of the above symptoms with reports of yellow rain attacks—that is, when yellow material is used these symptoms appear; other agents may give rise to other symptoms. Although it is possible to exhibit one or even several of these symptoms associated with traditional chemical warfare agents, no expert has been able to fit the sequence, severity, and consistency with any of them. In many cases, victims and observers were examined, histories taken, and interviews conducted by several health professionals weeks apart. Remarkable consistency has been observed.

From the beginning of the yellow rain episodes in 1975, autopsies occasionally have been reported anecdotally. Some have been done inexpertly, some by nonphysicians, and some were performed on animals rather than on human victims. However, the consistency of the early reported “putrefaction” or “rottenness” of the digestive tract within 12-48 hours after death led many forensic medical experts to suspect that one effect of the poison—whatever it was—was to cause necrosis (cell death) of rapidly dividing mucosa (mucous membranes), especially in the stomach and upper small intestine. Other autopsy findings included hyperemia (engorgement with blood) of digestive mucosal linings and remarkably intense congestion and swelling in the lungs, liver, spleen, and sometimes the kidneys. These and other findings often led experts in toxicology and pathology, on the basis of clinical and pathological data alone, to suggest mycotoxin or even trichothecene intoxication.

Trichothecene effects have been reported in the forensic, oncological, and toxicological literature for several years. Unpublished findings often have been discussed in symposiums. In several dozen cases, toxic effects in humans and

animals have been carefully recorded, and they match those of yellow rain with good precision (see Table C-1). *There are no additional signs or effects of known trichothecene intoxication not frequently reported by victims, nor are there any reported yellow rain symptoms that cannot be explained by the effects of the four specific trichothecene toxins found in the samples.*

There are no significant medical differences in the reporting from Laos and Kampuchea. Although the timing and delivery systems have sometimes varied, the effects of the chemical agent, clinically and pathologically, are identical. In some cases, a series of blood samples from Kampuchean victims also showed a trend toward leukopenia (reduction in the number of white blood cells) and the presence of a trichothecene metabolite (HT-2) consistent with trichothecene intoxication (see Annex D). Dose-response effects that were observed and routes of administration were both consistent with effects of trichothecenes.

An early hypothesis (1978-79) was that a significant number of deaths, especially in Laos, could be explained by the heavy use of riot-control agents such as CS, CN, DM, and agents which cause itching and/or blistering. This hypothesis was rejected quickly on two grounds. First, trace contaminant analysis failed to show the presence of any of these compounds in samples; several samples did, however, contain a trichothecene precursor. Second, contrary to commonly held views, the epidemiology of diseases endemic to the central highlands of Laos and the public health situation of the H'Mong do not support the view of malnourished, disease-ridden, and weak persons who would succumb easily to riot-control agents. Also, many studies have shown the opposite: a relatively low incidence of pulmonary disease, lower than what could otherwise account for certain effects; better nutritional states than could otherwise account for death in 10 days to 2 weeks from water loss (dehydration) and calorie depletion; and a death rate of nearly zero from causes other than infection, old age, and trauma.

### Afghanistan

Some deaths associated with bleeding have been described in the accounts from Afghanistan. In one set of cases, a physician examined persons who had been exposed to sublethal doses of a yellow smoke/black smoke combination attack and one man near death after a series of attacks. Hemoptysis (nasal

**TABLE C-1**

### Comparison of Reported "Yellow Rain" Effects With Known Trichothecene Effects

#### Yellow Rain Reports\*

1. Nausea, vomiting—severe, immediate
2. "Falling down, world turning"
3. "Burning of skin"—small blisters
4. "Shaking all over, flopping like fish out of water"
5. "Bleeding eyes"
6. "Pounding" chest, rapid heartbeat, weakness
7. Severe pain in center of chest
8. Sleepiness, "not able to talk"
9. Bleeding gums and profuse salivation
10. "Can't breathe"
11. "Skin and body hot with cold"
12. Diarrhea with blood
13. Loss of appetite, inability to eat
14. Bleeding into skin and fingernails
15. Drop in white blood cell count
16. "Rotten esophagus, stomach, intestines; soft spleen and liver"
17. Swelling of all organs

#### Effects of Trichothecenes

1. Nausea, vomiting—severe, immediate
2. Dizziness
3. Generalized erythema with a burning sensation of skin
4. Ataxia (failure of muscular coordination), occasional tremors and convulsions
5. Congestion of the sclera (white outer coat of eyeball) and blood in tears
6. Hypotension (abnormally low blood pressure) with secondary rise in heart rate
7. Angina (substernal chest pain)
8. Somnolence, central nervous system symptoms
9. Stomatitis (inflammation of oral mucous membranes) and pyalism (excessive salivation)
10. Shortness of breath
11. Fever and chills
12. Diarrhea with blood
13. Anorexia
14. Thrombocytopenia (decrease in number of platelets, white blood cells involved in clotting of blood) and purpura (skin discoloration caused by hemorrhage into tissues)
15. Leukopenia and anemia
16. Rapid necrosis of linings of gastrointestinal tract; lymphoid necrosis in spleen and liver
17. Congestion of all organs

\* Effects are immediate at levels near to or above a rough estimate of 500-1,000 mg total body burden for an adult. Although inhalation data are pending, the levels are consistent with reported lethal and sublethal doses. Trichothecenes in combination, when directly ingested or inhaled, or in purified form, are more toxic in lower concentrations, and the order of signs and symptoms and timing varies.

bleeding)—but not hematemesis (bleeding from the gastrointestinal tract)—was reported in about half of these cases.

Several features of at least one of the chemical agents—an incapacitant—used in Afghanistan defy explanation at this time. One possibility is that the agent(s) are highly selective for the central nervous system rather than the autonomic nervous system. As yet, no good candidate agent has been identified which will selectively inhibit the central nervous system so as to cause unconsciousness for several hours. Another finding has been the presence of a der-

mal anaesthesia, affecting only exposed areas of skin.

### Postattack Medical Survey

There is evidence that after some attacks in Laos and Afghanistan, Lao Communist or Soviet forces entered the attack zones to conduct surveys. Several reports indicate that survivors from a toxin attack on a Lao village were taken several kilometers from the village and injected with a small volume of a clear solution said by their captors to be a "new" medicine to assess the gas. The injections, given intramuscularly in the upper arm, reportedly did nothing to

alleviate the weakness, nausea, vomiting, or diarrhea suffered by the survivors. One victim reported the drug caused an immediate sensation of warmth throughout his body. Only the use of opium later eased the discomfort. It is probable that this procedure was a test either of a new antidote or of a drug developed to reduce incapacitation from the nausea and vomiting.

Similarly, in a few cases in Afghanistan, Soviet troops reportedly disembarked from helicopters or armored personnel carriers at the edge of an attack site. Three or four, dressed in full anti-contamination gear, walked among the dead, examined the corpses and, opening them with a crude incision, examined the organs in the abdominal and thoracic cavities. In one case, a solution was poured into the incision. When the corpses were later recovered by the *mujahidin*, the body cavity contents had been destroyed beyond recognition. These and a few additional reports support the hypothesis that the perpetrators of some of the attacks were interested in studying aftereffects, lethality, or some other quasi-experimental aspect of the use of a new chemical weapon. Recent indications from Afghanistan indicate that one purpose of the field surveys and body examinations is to determine levels of toxic materials still present in the attack zone before Soviet troops occupy it.

## ANNEX D

### ANALYSIS AND REVIEW OF TRICHOHECENE TOXINS

#### Sample Analyses for Trichothecenes

**The Trichothecene Hypothesis.** Since 1975, the U.S. Government has received remarkably consistent reports detailing chemical attacks in Southeast Asia. Some of these reports described the use of lethal agents which produced symptoms that could not be correlated with those produced by known or traditionally recognized chemical warfare agents or combinations of them (see Table D-1). It is readily apparent that the symptoms most frequently described in Laos and Kampuchea correspond most closely with those produced by a group of mycotoxins—the trichothecenes. A review of the scientific literature revealed not only that these compounds had physical and chemical properties indicating potential as chemical agents but also that they were the subject of intensive investigation by Soviet scientists at institutes previously linked with chemical and biological warfare research. In the fall of

1980, the trichothecenes were added to the list of agents suspected to have been used in Southeast Asia and Afghanistan. Other candidates under consideration included phosgene oxime, arsines, cyanogen chloride, nerve agents, riot-control agents, and combinations of these agents.

Many samples from chemical attacks in Laos and Kampuchea were examined at the U.S. Army's Chemical Systems Laboratory (CSL) for the presence of traditional chemical warfare agents and were reported to be negative. In March 1981, CSL reported the presence of an unusual compound ( $C_{15}H_{24}$ ) in the vapor analyses from several clothing and tissue samples taken from the victim of a chemical attack. The compound was closely related in structure to the simple trichothecenes. This finding sparked the request for analysis of all future samples for the presence of trichothecene mycotoxins.

**The Kampuchean Leaf and Stem Sample: The First Analysis for Trichothecenes.** On March 24, 1981, a number of samples were received from the U.S. Embassy in Bangkok. Two were reported to have been collected from the site of a chemical attack that occurred in the vicinity of TV 3391, an area just south of Phnom Mak Hoeun. A vegetation sample and a water sample were collected within 24 hours of the attack. Examination of bodies of victims of this attack by medical personnel revealed highly unusual degeneration of the mucosal lining of the gastrointestinal tract. The effects described paralleled those known to be produced by the trichothecenes. The samples were submitted to the Chemical Systems Laboratory for analysis for the presence of chemical warfare agents. With the exception of the unusual presence of high levels of CN-, Cl-, and F-ions, no evidence of known chemical warfare agents was found. An initial test for the trichothecenes by thin layer chromatography was inconclusive because of severe problems with interfering substances and the lack of appropriate standards.

The trichothecenes are difficult to detect even under ideal circumstances, and the presence of interfering substances in the sample may make identification and quantification by thin layer chromatography inconclusive. A review of the limitations and potentials of the analytical methods for trichothecenes led to the conclusion that the computerized gas chromatography/mass spectroscopy method in the selected ion-monitoring mode enabled precise identification and quantification of these compounds in complex mixtures. A comparison of the

currently available methods suitable for trichothecene analysis and an assessment of their utility and limitations is presented in Table E-3.

A portion of the leaf and stem sample was furnished to the U.S. Army Medical Intelligence and Information Agency for further analysis. This sample, a positive control sample to which T-2 toxin was added, and a negative control sample of similar vegetation were forwarded to Dr. Chester J. Mirocha of the Department of Plant Pathology, University of Minnesota. Dr. Mirocha was given no information concerning the history or content of the samples and was requested to analyze the three unknowns for the presence of trichothecene toxins using the best methods at his disposal.

The analysis involves a series of extractions followed by ferric gel separation, selected ion monitoring on a computerized gas chromatograph/mass spectrometer, and a full mass spectral scan for comparison with known standards. The methods used are among the most sensitive and specific for detection of these compounds; also, false positives are rare. Toxins can be identified by their mass spectra and quantified with a high degree of accuracy. The vegetation sample allegedly exposed to a chemical warfare agent was found to contain 109 parts per million (ppm) of nivalenol, 59.1 ppm of deoxynivalenol, and 3.15 ppm of T-2 toxin; each is a potent toxin of the trichothecene group. No trichothecenes were detected in the negative control sample, and 35 ppm of T-2 toxin were detected in the sample to which T-2 toxin had been added. It was Dr. Mirocha's assessment that a mixture of these particular toxins in the high levels detected could not have occurred as a result of natural contamination.

The possibility that the identified toxins were produced by natural fungal contamination was discounted on the basis of the climatic conditions required for production of T-2 toxin, the high levels of toxins detected, the unusual mixture of toxins found, and the results of surveys of Southeast Asia for the presence of these toxins. This conclusion was supported by the analysis of normal flora samples from Kampuchea described below.

**Analyses of Control Samples From Kampuchea for the Presence of Trichothecenes.** On September 20, 1981, the U.S. Army Medical Intelligence and Information Agency received nine control samples from U.S. Army personnel in Bangkok for the purpose of conducting laboratory analyses for background

TABLE D-1

## Symptoms of Chemical Attacks Reported in Laos, Kumpuchea, and Afghanistan

| Symptom                     | % of Reports<br>Mentioning<br>Symptom | Tricho-<br>thecenes | Nerve<br>Agents | Arsines | Phosgene<br>Oxime | Cyanogens | Incapacitant<br>(BZ) | Riot-<br>Control<br>Agents |
|-----------------------------|---------------------------------------|---------------------|-----------------|---------|-------------------|-----------|----------------------|----------------------------|
| <b>Laos</b>                 |                                       |                     |                 |         |                   |           |                      |                            |
| Multiple deaths             | 84.6                                  | X                   | X               | X       | —                 | X         | —                    | —                          |
| Vomiting                    | 71.4                                  | X                   | X               | X       | —                 | —         | —                    | X                          |
| Diarrhea                    | 53.1                                  | X                   | X               | X       | —                 | —         | —                    | —                          |
| Hemorrhage                  | 52.0                                  | X                   | —               | —       | X <sup>a</sup>    | —         | —                    | —                          |
| Breathing difficulty        | 47.95                                 | X                   | X               | X       | X                 | X         | X                    | X                          |
| Itching and skin irritation | 43.9                                  | X                   | —               | X       | X                 | —         | —                    | X                          |
| Nausea                      | 42.8                                  | X                   | X               | X       | —                 | —         | X                    | X                          |
| Animal death                | 41.8                                  | X                   | X               | X       | —                 | X         | —                    | —                          |
| Blurred vision              | 39.7                                  | X                   | X               | X       | X                 | X         | X                    | X                          |
| Headache                    | 36.7                                  | X                   | X               | —       | X                 | —         | X                    | X                          |
| Fatigue                     | 35.7                                  | X                   | X               | —       | —                 | —         | X                    | —                          |
| Nasal excretion             | 34.7                                  | X                   | X               | X       | X                 | —         | —                    | X                          |
| Rash or blisters            | 32.6                                  | X                   | —               | X       | X                 | —         | —                    | X                          |
| Tearing                     | 30.6                                  | X                   | X               | X       | X                 | X         | —                    | X                          |
| Coughing                    | 28.6                                  | X                   | X               | X       | X                 | X         | —                    | X                          |
| Effect on vegetation        | 26.5                                  | X                   | —               | X       | X                 | —         | —                    | —                          |
| Dizziness and vertigo       | 25.5                                  | X                   | X               | —       | —                 | X         | X                    | X                          |
| Facial edema                | 20.4                                  | X                   | —               | X       | X                 | —         | —                    | X                          |
| Thirst and dry mouth        | 20.4                                  | X                   | —               | —       | —                 | —         | X                    | —                          |
| Skin color change           | 16.3                                  | X                   | —               | —       | X                 | —         | —                    | —                          |
| Tachycardia                 | 12.3                                  | X                   | X               | —       | X                 | X         | X                    | X                          |
| Temporary blindness         | 9.18                                  | X                   | —               | X       | X                 | —         | X                    | X                          |
| Rapid loss of consciousness | 9.18                                  | X <sup>b</sup>      | X               | —       | —                 | X         | X                    | —                          |
| Salivation                  | 6.12                                  | X <sup>c</sup>      | X               | —       | —                 | —         | —                    | —                          |
| Hearing loss                | 5.1                                   | X                   | —               | —       | —                 | —         | —                    | —                          |
| Tremors or convulsions      | 4                                     | X                   | X               | —       | X                 | X         | —                    | —                          |
| Sweating                    | 3                                     | —                   | X               | —       | —                 | —         | —                    | —                          |
| Paralysis                   | 3                                     | X                   | X               | —       | —                 | X         | —                    | —                          |
| Loss of appetite            | 3                                     | X                   | X               | X       | —                 | —         | —                    | —                          |
| Frequent urination          | 2                                     | X                   | X               | —       | —                 | —         | —                    | —                          |

Continued on p. 25

**Note:** This table is a compilation relating the signs and symptoms reported in the three countries to symptoms associated with certain chemical agents. The frequency with which a particular symptom was reported is expressed as a percentage of the total number of attacks.

levels of trichothecene toxins. The samples were collected from an area near TV 3391 that had not been subjected to any reported chemical attacks. The samples were collected by U.S. personnel under instructions to reproduce the sampling conditions, handling, packaging, and transfer conditions of the original sample as closely as possible. The same species of plant was sampled, and four other vegetation samples also were collected. A water sample and two soil samples were recovered. Corn and rice samples from the area also were taken. These grains provided an ideal substrate for growth of toxin-producing fungi and would, therefore, be a sensi-

tive indicator of any natural occurrence. The nine samples were forwarded under code to Dr. Mirocha for trichothecene analysis. A portion of each sample also was submitted to Chemical Systems Laboratory for background determinations of CN-, Cl-, and F-levels. No trichothecenes were detected in any of these samples, indicating that nivalenol, deoxynivalenol, T-2, and diacetoxyscirpenol are not prevalent in the geographical area from which the alleged chemical warfare-exposed sample was collected. The appearance of these trichothecenes in high levels and unique combinations in a sample associated with a chemical attack—which produced symptoms typical of trichothecene exposure—indicates

that these toxins may have been used as chemical weapons. This conclusion is further supported by the evidence provided by analysis of additional alleged chemical warfare samples from Laos and Kampuchea as described below.

**Analysis of Additional Chemical Warfare Samples From Laos and Kampuchea for the Presence of Trichothecenes.** The U.S. Army Medical Intelligence and Information Agency received from the Chemical Systems Laboratory three additional suspected chemical warfare samples for analysis for trichothecenes. The first sample consisted of 10 ml of water taken from the same chemi-

TABLE D-1 (continued)

## Symptoms of Chemical Attacks Reported in Laos, Kumpuchea, and Afghanistan

| Symptom                     | % of Reports<br>Mentioning<br>Symptom | Tricho-<br>thecenes | Nerve<br>Agents | Arsines | Phosgene<br>Oxime | Cyanogens | Incepecitent<br>(BZ) | Riot-<br>Control<br>Agents |
|-----------------------------|---------------------------------------|---------------------|-----------------|---------|-------------------|-----------|----------------------|----------------------------|
| <b>Kampuchea</b>            |                                       |                     |                 |         |                   |           |                      |                            |
| Multiple deaths             | 72.4                                  | X                   | X               | X       | —                 | X         | —                    | —                          |
| Hemorrhage                  | 62.06                                 | X                   | —               | —       | X <sup>d</sup>    | —         | —                    | —                          |
| Dizziness and vertigo       | 51.7                                  | X                   | X               | —       | —                 | X         | X                    | X                          |
| Vomiting                    | 41.3                                  | X                   | X               | X       | —                 | —         | —                    | X                          |
| Nausea                      | 34.5                                  | X                   | X               | X       | —                 | —         | X                    | X                          |
| Skin irritation             | 27.6                                  | X                   | —               | X       | X                 | —         | —                    | X                          |
| Rapid loss of consciousness | 24.1                                  | X <sup>b</sup>      | X               | —       | —                 | X         | X                    | —                          |
| Fever                       | 20.68                                 | X                   | —               | —       | —                 | —         | —                    | —                          |
| Headache                    | 17.2                                  | X                   | X               | —       | X                 | —         | X                    | X                          |
| Tearing                     | 13.8                                  | X                   | X               | X       | X                 | X         | X                    | X                          |
| Breathing difficulty        | 13.8                                  | X                   | X               | X       | X                 | X         | X                    | X                          |
| Fatigue                     | 13.8                                  | X                   | X               | —       | —                 | —         | X                    | —                          |
| Paralysis                   | 10.3                                  | X                   | X               | —       | —                 | X         | —                    | —                          |
| Numbness                    | 6.9                                   | X                   | X               | —       | —                 | X         | X                    | —                          |
| Blurred vision              | 6.9                                   | X                   | X               | X       | X                 | X         | X                    | X                          |
| Dry throat and thirst       | 6.9                                   | X                   | —               | —       | —                 | —         | X                    | —                          |
| Edema                       | 6.9                                   | X                   | —               | X       | X                 | —         | —                    | —                          |
| Salivation                  | 3.4                                   | X <sup>c</sup>      | X               | —       | —                 | —         | —                    | —                          |
| Vegetation affected         | 3.4                                   | X                   | —               | X       | —                 | —         | —                    | —                          |
| Diarrhea                    | 3.4                                   | X                   | X               | X       | —                 | —         | —                    | —                          |
| Cough                       | 3.4                                   | X                   | —               | X       | X                 | X         | X                    | X                          |
| Nasal discharge             | 3.4                                   | X                   | X               | X       | X                 | —         | —                    | X                          |
| Rash or blister             | 3.4                                   | X                   | —               | X       | X                 | —         | —                    | X                          |
| Chills                      | 3.4                                   | X                   | ?               | —       | —                 | —         | —                    | —                          |
| Hearing loss                | 3.4                                   | X                   | —               | —       | —                 | —         | —                    | —                          |
| <b>Afghanistan</b>          |                                       |                     |                 |         |                   |           |                      |                            |
| Rapid loss of consciousness | 47.9                                  | X <sup>b</sup>      | X               | —       | —                 | X         | X                    | —                          |
| Skin irritation and itching | 31.5                                  | X                   | —               | X       | X                 | —         | —                    | X                          |
| Multiple deaths             | 30.1                                  | X                   | X               | X       | —                 | X         | —                    | —                          |
| Nausea                      | 20.5                                  | X                   | X               | X       | —                 | —         | X                    | X                          |
| Vomiting                    | 19.1                                  | X                   | X               | X       | —                 | —         | —                    | X                          |
| Tearing                     | 17.8                                  | X                   | X               | X       | X                 | X         | —                    | X                          |
| Dizziness and vertigo       | 16.4                                  | X                   | X               | —       | —                 | X         | X                    | X                          |
| Blisters or rash            | 15                                    | X                   | —               | X       | X                 | —         | —                    | X                          |
| Difficulty breathing        | 13.7                                  | X                   | X               | X       | X                 | X         | X                    | X                          |
| Paralysis                   | 13.7                                  | X                   | X               | —       | —                 | X         | —                    | —                          |
| Headache                    | 12.3                                  | X                   | X               | —       | X                 | —         | X                    | X                          |
| Temporary blindness         | 8.2                                   | X                   | —               | X       | X                 | —         | X                    | X                          |
| Salivation                  | 6.8                                   | X <sup>c</sup>      | X               | —       | —                 | —         | —                    | —                          |
| Loss of appetite            | 6.8                                   | X                   | X               | X       | —                 | —         | —                    | —                          |
| Effects on vegetation       | 5.5                                   | X                   | —               | —       | —                 | —         | —                    | —                          |
| Fatigue                     | 5                                     | X                   | X               | —       | —                 | —         | X                    | —                          |
| Confusion                   | 4.1                                   | X                   | X               | —       | —                 | —         | X                    | —                          |
| Hemorrhage                  | 4.1                                   | X                   | —               | —       | X <sup>a</sup>    | —         | —                    | —                          |
| Change in skin color        | 2.8                                   | X                   | —               | —       | X                 | —         | —                    | —                          |
| Diarrhea                    | 2.8                                   | X                   | X               | X       | —                 | —         | —                    | —                          |
| Coughing                    | 1.3                                   | X                   | X               | X       | X                 | X         | X                    | X                          |

<sup>a</sup> Bloody frothing.<sup>b</sup> Only at very high doses.<sup>c</sup> Depending on which trichothecenes.<sup>d</sup> Blood flecked frothing.

cal attack site in Kampuchea as the leaf and stem sample previously examined. The second sample came from the site of a "yellow rain" attack occurring on March 13, 1981, in the village of Muong Cha (TF 9797) in the Phou Bia region of Laos. The agent was sprayed from a twin-engine propellor aircraft at about noon, local time. The falling substance was described as "like insect spray" and sounded like drizzling rain. Quite sticky at first, it soon dried to a powder. Symptoms described by victims included nausea, vomiting, and diarrhea. A sample of the agent scraped from the surface of a rock by a victim and carried into Thailand was turned over to U.S. Embassy personnel. The third sample was taken from the site of a "yellow rain" attack that occurred at 2:00 p.m. on April 2, 1981, at Ban Thong Hak (TF 9177). Twenty-four people reportedly died in this attack; there were 47 survivors. Symptoms included severe skin irritation and rash, nausea, vomiting, and bloody diarrhea. A survivor of the attack scraped this sample from the surface of a rock with a bamboo knife. Although the individual took precautions (that is, cloth mask), a severe skin rash and blisters developed.

These three samples were submitted to Dr. Mirocha for analysis. The water sample from Kampuchea contained 66 ppm of deoxynivalenol and a trace amount of diacetoxyscirpenol. A trace quantity of the second sample was screened as strong positive for trichothecenes. Further analysis of that sample confirmed the presence of high levels of T-2 toxin (150 ppm) and diacetoxyscirpenol (25 ppm). Nivalenol and deoxynivalenol may also be present but are being masked by interference from phthalate compounds (leached from the plastic packaging). An effort to modify the extraction process is being made in order to overcome the interference so that nivalenol and deoxynivalenol can be measured more easily. Interestingly, examination of the petroleum ether fraction from the sample revealed the presence of a yellow pigment almost identical to that previously identified by Dr. Mirocha in cultures of *Fusarium roseum*, indicating that the yellow powder probably consisted of the crude extract of a *Fusarium* culture.

There was little of the third sample contained in the vial received for testing. The quantity was too small to be weighed accurately, and inspection of the vial revealed only a small speck estimated to weigh much less than 0.1 mg. That speck contained 10 ng of diacetoxyscirpenol, a level equivalent to

100 ppm at the very least and probably much higher. The sample size was too small to allow adequate analysis for the other three trichothecenes of interest.

These results support the hypothesis that trichothecenes have been used as chemical warfare agents in Laos and Kampuchea. The presence of these high levels of trichothecene toxins in water and in yellow powder scraped from rocks argues against natural occurrence, since neither water nor rock is a suitable environment for growth of the fungi required to produce the toxins.

Differences between the analyses of the Kampuchean leaf and stem sample and the water sample collected from the same attack site raise additional questions. Failure to find T-2 toxin in the water sample is probably due to the relative insolubility of T-2 toxin in water. The presence of diacetoxyscirpenol in the water might be the result of biotransformation or breakdown of T-2, as they are so structurally similar, differing only in the substitution on carbon 8. While this hypothesis cannot be entirely ruled out, it is unlikely on the basis of known biotransformation of T-2 in the laboratory. The initial vegetation sample was not screened for diacetoxyscirpenol, although the mass spectra from the initial analysis will be reexamined for trace amounts of it.

The absence of nivalenol in the water sample is more difficult to explain because nivalenol is water soluble. The effect of environmental conditions and microorganisms on the stability of these compounds may vary widely for each of the specific compounds and may explain the analytical results. Further scientific investigation of these factors is needed.

#### Analysis of Blood Samples From Chemical Attack Victims

Blood samples drawn from victims of recent chemical attacks in Kampuchea have been received by the U.S. Army Medical Intelligence and Information Agency for analysis for indications of trichothecene exposure. Little is known concerning the rate of metabolism of trichothecenes in humans; it is difficult, therefore, to estimate the probability of detecting trichothecenes or their metabolites in blood samples. T-2 is rapidly cleared from the blood in animals, and 25% of the total dose is excreted within 24 hours after exposure; it is unlikely that trichothecenes could be detected unless blood samples were obtained within 24-48 hours after an attack. Other blood parameters are affected by

the trichothecenes, however, and may prove to be useful markers. The trichothecenes induce a severe leukopenia (decrease in white cell count) which can persist for several weeks following exposure. In addition, the trichothecenes affect some liver and kidney function marker enzymes which can be monitored in the blood.

On October 11, 1981, four whole blood samples and four blood smears were received from the U.S. Embassy in Bangkok. The blood was drawn from four Khmer Rouge soldiers on October 7, 1981 at a Khmer Rouge hospital inside Kampuchea. Detailed medical histories as well as descriptions of the attack were recorded on each individual from whom a blood sample was taken. All four men were victims of a gas attack occurring near Takong on September 19, 1981. Symptoms experienced included vomiting, blurred vision, bloody diarrhea, difficult breathing, dry throat, loss of consciousness, frontal headache, tachycardia, and facial edema. Unfortunately, the samples could not be refrigerated until 48 hours after collection. Thus, it was impossible to obtain data concerning white cell counts and blood chemistry. The four whole blood samples were submitted to Dr. Mirocha for analysis for trichothecene metabolites because of the possibility, admittedly remote, that some of the metabolites might bind to blood proteins and might still be detectable even 3 weeks after an attack.

On October 22, 1981, additional blood samples were received. These had been drawn from nine victims from the September 19 attack and from four control individuals of similar age and background who had not been exposed to a chemical attack. The samples had been properly refrigerated and were accompanied by complete and detailed medical histories taken by trained medical personnel who examined the individuals. Included in the package were blood smears and heparinized and nonheparinized samples from each individual. The samples were submitted for blood assays to the U.S. Army Medical Research Institute of Infectious Diseases.

The above results show no statistically significant differences between exposed and control groups (students T-test). In eight individuals exposed to a chemical agent, a trend toward depressed white cell counts was observed. Such an observation would be compatible with the clinical picture of toxin exposure; however, it is also compatible with a number of other medical problems, and a larger control sample would

be required before such results could be adequately interpreted. Abnormal liver and kidney functions were not indicated by these data.

Portions of the blood samples were analyzed by Dr. Mirocha for the presence of trichothecenes and/or trichothecene metabolites. The results of the analyses are consistent with trichothecene exposure in at least two of the gassing victims and tend to support the hypothesis that a trichothecene-based agent was used in this attack.

Using the selected ion-monitoring gas chromatography/mass spectroscopy analysis technique, Dr. Mirocha was able to identify tentatively a metabolite of T-2 toxin (that is, HT-2) in the blood of two alleged victims. The compound was identified on the basis of its selected ion masses and gas chromatographic retention times.

The tentative identification of HT-2 in the blood of two victims, and the trend toward depressed white cell counts in these same victims, cannot be taken as conclusive scientific proof of toxin exposure because the trace amount of the compound present precluded unequivocal identification and quantification and because many other medical problems in addition to toxin exposure can cause a decrease in white cell counts. It is interesting to note that the individual who showed the greatest amount of the compound tentatively identified as HT-2 in his blood reportedly received the greatest exposure to the agent. He was exposed to contaminated water for more than 30 minutes and was the only victim who fell down in the water and actually swallowed some of it. However, the description by victims of symptoms correlating exactly with those associated with trichothecene poisoning provides strong circumstantial evidence that trichothecenes were used as chemical agents in yet another chemical attack in Southeast Asia.

Trichothecenes have been identified previously in environmental samples taken from several other chemical attacks in Laos and Kampuchea. Analysis of control vegetation, water, soil, corn, and rice samples from these areas, as well as reviews of published scientific literature, indicates that the particular toxins that have previously been identified are not known to occur naturally in the combinations found and at the levels detected in Southeast Asia. The latest analysis results contribute another piece of evidence to the growing body of data supporting the charge that trichothecenes have been used as chemical/biological agents in Southeast Asia.

## ANNEX E

### OVERVIEW OF NATURAL OCCURRENCE AND SIGNIFICANT PROPERTIES OF TRICHTHOCENES

#### Historical Trichothecene Mycotoxins

The trichothecenes are members of a large group of naturally occurring toxins known as mycotoxins. The word "mycotoxin" is derived from the Greek "mykes" meaning fungus and the Latin "toxicum" meaning poison. It refers to a metabolite produced by a mold that is toxic to man and animals. Mycotoxins have been described as the "neglected diseases," and before 1960 English-language literature concerning the diseases caused by mycotoxins was scarce. Soviet scientists have been involved in research with some of these compounds for almost 30 years longer than their Western counterparts. The Soviet Union has had serious problems with mycotoxin contamination of food and has suffered several severe outbreaks of disease in humans. The first comprehensive studies of mycotoxin diseases were conducted in the Soviet Union in the late 1930s.

Since the 1940s, the group of mycotoxins figuring most prominently in Soviet scientific literature are the trichothecenes, a class of chemically related, biologically active fungal metabolites produced primarily by various species of *Fusarium*. Table E-1 lists some of the toxins in this group and producing fungi. The fungi are well-known plant pathogens that frequently invade many agricultural products.

Trichothecene toxins, perhaps more than any other mycotoxins, have been associated with acute disease in humans. Most of the human intoxications have occurred in the Soviet Union (Table E-2). The earliest recognized outbreak occurred in 1891 in the Ussuri district of eastern Siberia. Humans who consumed contaminated grain exhibited headache, chills, nausea, vomiting, vertigo, and visual disturbances. Dogs, horses, pigs, and domestic fowls reportedly were affected.

The most extensive mycotoxicosis outbreak reported to have caused multiple fatalities in man also occurred in the Soviet Union. In 1944, 30% of the population of Orenburg district, near Siberia, was affected by alimentary toxic aleukia (ATA), a disease later shown to be caused by ingestion of trichothecene toxins. More than 10% of the entire

population of the district died of the disease. Many other outbreaks of ATA occurred in the Soviet Union, mainly during the 1942-47 period. The contamination was traced to overwintered millet, wheat, and barley infected with *Fusarium*. Symptoms of the disease included vomiting, skin inflammation, multiple hemorrhaging (especially of the lung and gastrointestinal tissue), diarrhea, leukopenia, and suppression of bone marrow activity.

In 1939, Premier Joseph Stalin dispatched Nikita Khrushchev to the Ukraine to organize and improve agricultural operations and to identify the disease causing the deaths of many horses and cattle. The problem was traced to hay and straw contaminated with *Stachybotrys atra*. The disease, later referred to as stachybotryotoxicosis, occurred after ingestion or contact with the contaminated grain. Symptoms included ulcerative dermatitis, peroral dermatitis, blood dyscrasias, hemorrhagic syndromes, abortion, and death. The greatest economic impact was due to loss of horses, although cattle, sheep, poultry, and humans also were affected.

Other disease outbreaks in which similar symptoms were present occurred in 1958 and 1959 among horses and cattle in the Soviet Union and Eastern Europe; thousands of animals were lost. Other intoxications were reported later

#### Soviet Scientists Involved in Mycotoxin Research

- |  |                     |
|--|---------------------|
| A. Kh. Sarkisov—All Union Scientific Research Institute of Experimental Veterinary Science, Moscow | A. M. Kogan         |
| V. I. Bilay (also spelled Bilai)—Ukrainian S.S.R. Institute of Microbiology and Virology, Kiev     | D. T. Martynenko    |
| V. A. Tutelyan—U.S.S.R. Academy of Medical Sciences Nutrition Institute, Moscow                    | N. A. Kostyunina    |
| M. A. Akhmeteli—U.S.S.R. Academy of Medical Sciences Institute of Epidemiology and Microbiology    | V. V. Yerinakov     |
| L. Ye. Olifson   | I. A. Kurmanov      |
| M. F. Nesterin   | V. V. Semenov       |
| K. Z. Salomatina   | Z. K. Bystryakova   |
| Ye. P. Kozhevnikov   | Z. Z. Orlova        |
| N. D. Osadchaya  | L. S. L'vova        |
| L. F. Mikhaylova   | L. I. Lozbina       |
| Sh. M. Kenina  | T. A. Shevtsova     |
| V. L. Kartashova   | I. Yu. Makedon      |
| L. R. Filonova   | N. S. Proskuryakova |
| T. Ye. Tolcheyeva  | A. V. Borovkov      |
| Kn. A. Dzhlavayan  | M. N. Nazypov       |
| I. S. Yelistratov  | L. I. Lozbina       |
| N. S. Tishkova   | M. S. Marova        |
| V. I. Kaplun   | P. A. Il'in         |
| Ye. P. Kozhevnikova  |                     |
| S. M. Gubkin   |                     |
| L. I. Il'ina   |                     |

in Japan, Europe, the Soviet Union, and the United States, affecting various domestic animals and—in the case of “red mold toxicosis”—man. All of these diseases have now been shown to be due to ingestion of trichothecenes rather than to an infectious agent. In earlier outbreaks, the levels of toxin present in the contaminated grain were not measured; however, the levels of nivalenol and/or deoxynivalenol measured in toxic grains implicated in more recent outbreaks (i.e., “moldy corn toxicosis” and “red mold toxicosis”) typically were between 2 and 8 ppm.

#### Natural Occurrence of Trichothecene Mycotoxins

Publications concerning the occurrence of trichothecenes are relatively scarce because of the lack of convenient detection methods and the complexity of the trichothecene family of compounds. Only recently have scientists developed methods capable of distinguishing between close structural derivatives and accurately quantifying the levels of toxin present (see Table E-3 for comparison of analytical methods). Extreme care must be taken when reviewing the scientific literature on natural occurrence of these compounds because erroneous conclusions can be drawn on the basis of results obtained with inadequate analytical techniques. Misidentification of compounds and gross overestimation of concentrations have occurred using techniques such as thin layer chromatography.

Table E-4 lists the reports of natural occurrence of T-2 toxin, diacetoxyscirpenol, and nivalenol that were obtained from a literature search of more than 3,000 citations concerned with trichothecene toxins. Levels that are questionable on the basis of techniques used are indicated. It is immediately apparent that the levels of toxins found in the various samples from Laos and Kampuchea are highly unusual, even if one accepts the questionable reports in Table E-4 as valid. The levels of these toxins (150 ppm of T-2 toxin, 109 ppm of nivalenol, more than 100 ppm of diacetoxyscirpenol, and 66 ppm of deoxynivalenol) are markedly higher than those reported to occur in nature. It should also be noted that the incidences recorded in Table E-4 concern levels of toxin produced when *Fusarium* is growing on its ideal substrate, while the Laos

TABLE E-1

#### Trichothecene-Producing Fungi

| Type           | T-2 Type                   | Nivalenol-Type        | Meccrocyclic                         |
|----------------|----------------------------|-----------------------|--------------------------------------|
| Trichothecenes | T-2 Toxin                  | Nivalenol             | Roridins                             |
|                | HT-2 Toxin                 | Monoacetyl-Nivalenol  | Veirucarins                          |
|                | Diacetoxyscirpenol         | Diacetyl-Nivalenol    | Satratoxins                          |
|                | Neosolaniol                | Deoxynivalenol        | Vertisporin                          |
| Fungus         | <i>F. tricinctum</i>       | <i>F. nivale</i>      | <i>Myrothecium verrucaria</i>        |
|                | <i>F. roseum</i>           | <i>F. opisphaeria</i> |                                      |
|                |                            |                       | <i>M. roridum</i>                    |
|                | <i>F. equiseti</i>         | <i>F. roseum</i>      |                                      |
|                | <i>F. sporotrichioides</i> |                       | <i>Stachybotrys atra</i>             |
|                |                            |                       | <i>Verticimonosporium diffractum</i> |
|                | <i>F. lateritium</i>       |                       |                                      |
|                | <i>F. poae</i>             |                       |                                      |
|                | <i>F. solani</i>           |                       |                                      |
|                | <i>F. rigidusculum</i>     |                       |                                      |
|                | <i>F. semitectum</i>       |                       |                                      |

TABLE E-2

#### Historical Trichothecene Mycotoxicoses

| Toxicosis                  | Districts and Affected Species        | Symptoms   |
|----------------------------|---------------------------------------|--|
| “Tausalgetreide” Toxicosis | U.S.S.R.: man, farm animals           | Headache, nausea, vomiting, vertigo, chills, visual disturbances               |
| Alimentary toxic aleukia   | U.S.S.R.: man, horse, pig             | Vomiting, diarrhea, multiple hemorrhage, skin inflammation, leukopenia, angina |
| Stachybotryotoxicosis      | U.S.S.R., Europe: horse               | Shock, stomatitis, hemorrhage, dermal necrosis, nervous disorders              |
| Bean-hull toxicosis        | Japan: horse                          | Convulsion, cyclic movement  |
| Dendrodochiotoxicosis      | U.S.S.R., Europe: horse               | Skin inflammation, hemorrhage  |
| Moldy corn toxicosis       | United States: pig, cow               | Emesis, hemorrhage   |
| Red mold toxicosis         | Japan, U.S.S.R.: man, horse, pig, cow | Vomiting, diarrhea, congestion and hemorrhage of lung and intestine            |

and Kampuchea samples were taken from surfaces—rocks and water—that would be extremely unlikely to support *Fusaria* growth and toxin production. Higher levels of toxin production can, of course, be induced when the mold species is grown in pure culture under ideal laboratory conditions; for instance, the Soviets have succeeded in producing 4 grams of T-2 per kilogram of sub-

strate. In a natural environment, however, the *Fusaria* species cannot compete well with other molds such as species of *Aspergillus* and *Penicillium*, and levels of toxin produced are orders of magnitude lower.

The conclusion that the levels of toxins found in the Southeast Asia samples could have occurred only by means of an unnatural mechanism is also strengthened by surveys of the area conducted

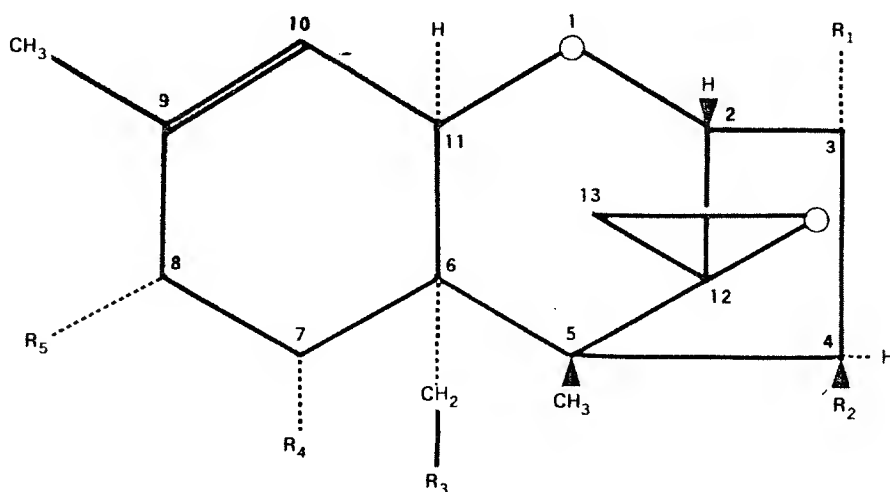
by various researchers. Surveys of the toxigenic fungi and mycotoxins naturally present in Southeast Asia conducted by the Mahidol University in Bangkok and the Massachusetts Institute of Technology have not revealed the presence of T-2, nivalenol, deoxynivalenol, or diacetoxyscirpenol, although other mycotoxins such as aflatoxin were identified. These results were confirmed by our analysis, using our own methodology, of normal flora samples of vegetation, soil, water, corn, and rice from Kampuchea that failed to reveal the presence of trichothecenes.

Skeptics have formulated theoretical explanations for the analytical results to support a hypothesis of natural occurrence of these toxins. It was postulated that the trichothecenes found were absorbed through the roots of a plant, translocated to the leaves, and exuded and washed onto the surface of a rock and into water where they were found. A 1981 publication by Jarvis et al. reported a Brazilian shrub that appeared to absorb, translocate, and chemically alter a macrocyclic trichothecene produced by soil fungi. While this citation is used to support a hypothetical mode for natural deposition in Southeast Asia, it should be noted that the plant reported in this publication did not exude the toxin, that the toxin was extremely phytotoxic to all other plants assessed, and that the plant was not capable of de novo trichothecene synthesis. No other trichothecenes have been found to be absorbed and translocated in any other plant in this manner. Control samples of soil and vegetation from Southeast Asia do not support endemic presence of these toxins. The appearance of these particular trichothecene toxins in these high levels in environments generally inhospitable to their formation cannot reasonably be attributed to a natural contamination.

### Chemical and Physical Properties of the Trichothecenes

When considering the suitability of trichothecenes as agents, factors such as stability, solubility, and ease of production must be considered. The general structure for the trichothecene group is shown in Figure E-1. There are more than 40 currently known, naturally occurring, 12 to 13 epoxytrichothecenes. The R groups may be hydroxyls, acylated hydroxyl groups or esters. The R group for the toxins detected in the sample is shown below the general structure. All of the compounds have in common an olefinic double bond at car-

FIGURE E-1  
General Structure of Trichothecenes



| T <sub>2</sub> Toxin   | Nivalenol          | Deoxynivalenol     |
|--|--------------------|--------------------|
| R <sub>1</sub> =OH   | R <sub>1</sub> =CH | R <sub>1</sub> =OH |
| R <sub>2</sub> =OAc  | R <sub>2</sub> =CH | R <sub>2</sub> =H  |
| R <sub>3</sub> =OAc  | R <sub>3</sub> =OH | R <sub>3</sub> =OH |
| R <sub>4</sub> =H  | R <sub>4</sub> =OH | R <sub>4</sub> =OH |
| R <sub>5</sub> =OCOCH <sub>2</sub> CH(CH <sub>3</sub> ) <sub>2</sub> | R <sub>5</sub> =O  | R <sub>5</sub> =O  |

bon atoms 9 and 10 and an epoxy group at carbon atoms 12 and 13. These compounds are stable, especially in the solid form. They may be stored for years at room temperature with no loss of activity. They are heat stable with no loss of activity noted after heating for 1 hour at 100° centigrade. The solubility depends on the R groups; highly hydroxylated derivatives are more water soluble. The compounds are also quite stable in solution. Detoxification can be accomplished

by treatment with strong mineral acid, which will open the 12 to 13 epoxide bond and abolish all biological activity. Most of the toxins are well absorbed through mucous membranes and some through skin; this property is also a function of the R group.

Some of these compounds have been synthesized chemically; however, biosynthesis employing *Fusarium* species is the most effective way to produce large quantities. In a preliminary search of recent Soviet literature, 50 articles dealing

TABLE E-3

## Physicochemical Methods for Detection of Trichothecenes in Feedstuffs

| Method  | Trichothecenes Detected            | Detection Limits                          | Required Standards                | Use and Limitation  |
|---|------------------------------------|---|-----------------------------------|---|
| Thin-layer chromatography 1-dimension                         | All                                | 0.1 microgram/spot ( $H_2SO_4$ )          | Reference Standard                | Qualitative<br>Interference<br>Not confirmatory                         |
| Thin-layer chromatography 2-dimension                         | All                                | 0.1-1.0 microgram/spot ( $H_2SO_4$ )      | Reference Standard                | Qualitative<br>Less interference<br>Confirmatory                        |
| Gas-liquid chromatography                                     | Nonhydroxylated or TMS derivatives | 0.03-0.05 microgram/microliter injection  | Reference Standard                | Quantitative<br>Monoglyceride interference<br>Equivocal identification  |
| Gas chromatography/mass spectrometry-normal scanning mode     | TMS derivatives                    | 0.02-0.05 microgram/microliter injection  | Reference Standard or Spectrogram | Semiquantitative<br>Less interference<br>Unequivocal identification     |
| Gas chromatography/mass spectrometry-selection ion monitoring | TMS derivatives                    | 0.007-0.02 microgram/microliter injection | Reference Standard or Spectrogram | Quantitative<br>Best for complex mixtures<br>Unequivocal identification |
| Nuclear-magnetic-resonance                                    | All                                | —   | Reference Standard or Spectrogram | Confirmatory<br>Purified toxin structure elucidation                    |
| Radio-immunoassay (developmental stage)                       | T-2 toxin                          | 1-20 nanogram                             | Rabbit anti-T-2 toxin anti-body   | Sensitive<br>Low interference   |
|   |                                    |   | HT-2 toxin                        | Relative structural specificity   |

with the trichothecenes were reviewed. Of these, 22 dealt with defining optimum conditions for biosynthesis of the compounds. N.A. Kostyunina has reported production of T-2 toxin at levels of 4 grams per kilogram of substrate (normally wheat grain or rice). Many industrial microbiology plants have been identified in the Soviet Union. Some are involved in production of single-cell protein for fodder additives, others produce antibiotics, and the function of still others is unknown. *Fusaria* are produced in the Soviet Union at a facility long reported in the open literature as being a suspected biological warfare agent production and storage facility. This facility, Berdsk Chemical Works, is near the science city of Novosibirsk in Siberia. The only difference between an antibiotic and mycotoxin is their target specificity. Both are produced by fungi, but the mycotoxins are relatively more

toxic to man than to microorganisms. Mycotoxins can be produced in good yield employing the same techniques used to produce some antibiotics. Thus, it may be concluded that the Soviets could produce trichothecenes in large amounts. They produce an antibiotic that is a trichothecene derivative, which would provide an ideal cover for agent production facilities.

#### Medical Effects of the Trichothecenes in Humans

The most prominent symptoms associated with trichothecene poisoning are listed in Table E-2. Striking among these is the rapid onset of vomiting, along with severe itching and tingling of the skin. Hemorrhage of the mucous membranes and bloody diarrhea follow. The symptoms shown in Table E-2 are similar to those reported by victims of trichothecene attacks in Laos, Kampu-

chea, and Afghanistan. The correlation is striking.

The  $LD_{50}$ 's (dose required to produce death in 50% of a test population) of the trichothecenes in laboratory animals range from 0.1 mg/kg to greater than 1,000 mg/kg, depending on the particular toxin, species, and route of exposure. The  $LD_{50}$  of T-2 toxin in a cat is 0.5 mg/kg. However, the  $ED_{50}$  (dose required to produce a desired physiological effect in 50% of a test population) is much lower. The  $ED_{50}$  to produce a vomiting reaction is 0.1 mg/kg; for skin irritation it is in the tenths of microgram range.

Most of the data concerning the toxicological effects of the trichothecenes are derived from animal data in which pure compounds were administered by oral, subcutaneous, intraperitoneal, or intravenous routes. Unfortunately, there are no reports concerning the effects of inhalation of mixtures of the compounds. Therefore, it is difficult to speculate concerning the effects that would be expected in humans exposed to an aerosol of mixtures of these potent toxins. The most useful data concerning exposure in humans were obtained in a phase I clinical evaluation of anguidine (diacetoxyscirpenol) as an anticancer drug. Diacetoxyscirpenol was administered by intravenous infusion. Doses of 3 mg/m<sup>2</sup>/day caused immediate onset of nausea, vomiting, diarrhea, somnolence and/or mental confusion, fever, chills, a generalized erythema with a burning sensation, hypotension, dyspnea, stomatitis, hives, and ataxia. Because of the side effects, the treatment was discontinued. The properties which make the use of diacetoxyscirpenol potentially useful as an anticancer drug are the same as those responsible, in part, for its extreme toxicity. It and the other trichothecenes cause extensive damage to rapidly dividing cells such as tumor cells. Unfortunately, the cells of the lining of the gastrointestinal tract and bone marrow are also rapidly dividing, and the effects of the trichothecenes on these cells result in severe, rapid degeneration of these tissues. The compounds also have direct effects on the clotting factors in the blood (that is, a primary effect on Factor VII activity and a secondary effect on prothrombin), which result in excessive hemorrhage following trauma.

The other useful body of clinical data concerning the effects of trichothecenes in humans is drawn from descriptions of the course of the disease in the natural

TABLE E-4

## Spontaneous Occurrence of Trichothecene Mycotoxins

| Toxin  | Country      | Source          | Concentration<br>(parts per<br>million) | Reference <sup>a</sup> | FOOTNOTES   |
|--|--------------|-----------------|---|------------------------|---|
| T-2 Toxin  | U.S.         | Mixed feed      | 0.08 <sup>b</sup>                       | 15                     | <sup>a</sup> References:<br>1. Balzer <i>et al.</i> (1977)<br>2. Ciegler (1978)<br>3. Eppley <i>et al.</i> (1974)<br>4. Funnel (1979)<br>5. Ghosal <i>et al.</i> (1978)<br>6. Ghosal <i>et al.</i> (1977)<br>7. Hibbs <i>et al.</i> (1974)<br>8. Hsu <i>et al.</i> (1972)<br>9. Isshi <i>et al.</i> (1975)<br>10. Jemmail <i>et al.</i> (1978)<br>11. Marasas <i>et al.</i> (1977)<br>12. Miller (1976)<br>13. Mirocha (1979)<br>14. Mirocha (1979)<br>15. Mirocha <i>et al.</i> (1976)<br>16. Mirocha <i>et al.</i> (1979)<br>17. Mirocha <i>et al.</i> (1979)<br>18. Morooka <i>et al.</i> (1972)<br>19. Petrie <i>et al.</i> (1977)<br>20. Puls and Greenway <i>et al.</i> (1976)<br>21. Romer, T., Ralston Purina,<br>St. Louis, MO (personal<br>communication)<br>22. Rukmini and Bhat (1978)<br>23. Siegfried (1979)<br>24. Vesonder and Ciegler (1979)<br>25. Vesonder <i>et al.</i> (1976)<br>26. Vesonder <i>et al.</i> (1978)<br><sup>b</sup> Zearalenone (F-2 toxins) also detected<br>in the sample.<br><sup>c</sup> ND = toxin concentration was not deter-<br>mined.<br><sup>d</sup> Levels that are questionable on the<br>basis of techniques used. |
|  | U.K.         | Brewer's grains | ND <sup>c</sup>                         | 19                     |   |
|  | India        | Sweet corn      | 4 <sup>b, d</sup>                       | 5                      |   |
|  | Canada       | Corn            | ND                                      | 4                      |   |
|  | India        | Sorghum         | ND <sup>d</sup>                         | 22                     |   |
|  | Canada       | Barley          | 25 <sup>d</sup>                         | 20                     |   |
|  | India        | Safflower seed  | 3-5 <sup>d</sup>                        | 6                      |   |
|  | U.S.         | Corn stalks     | 0.11 <sup>b</sup>                       | 16                     |   |
|  | U.S.         | Feed supplement | ND                                      | 7                      |   |
|  | U.S.         | Corn            | 2                                       | 8                      |   |
|  | U.S.         | Mixed feed      | 0.3                                     | 14                     |   |
|  | France       | Corn            | 0.02 <sup>b</sup>                       | 10                     |   |
|  | U.S.         | Corn            | ND                                      | 2                      |   |
| Diacetoxy-<br>scirpenol                                | U.S.         | Mixed feed      | 0.5                                     | 15                     |   |
|  | U.S.         | Mixed feed      | 0.38                                    | 15                     |   |
|  | India        | Safflower seed  | 3-5 <sup>d</sup>                        | 6                      |   |
|  | India        | Sweet corn      | 14 <sup>d</sup>                         | 5                      |   |
|  | Germany      | Corn            | 31.5 <sup>d</sup>                       | 23                     |   |
|  | U.S.         | Corn            | 0.88                                    | 21                     |   |
| Deoxynivalenol   | U.S.         | Corn stalks     | 1.5 <sup>b</sup>                        | 16                     |   |
|  | U.S.         | Corn            | 1.8 <sup>b</sup>                        | 15                     |   |
|  | U.S.         | Corn            | 1.0 <sup>b</sup>                        | 15                     |   |
|  | U.S.         | Corn            | 0.1 <sup>b</sup>                        | 15                     |   |
|  | U.S.         | Mixed feed      | 0.04 <sup>b</sup>                       | 15                     |   |
|  | U.S.         | Mixed feed      | 1.0 <sup>b</sup>                        | 15                     |   |
|  | U.S.         | Mixed feed      | 1.0 <sup>b</sup>                        | 15                     |   |
|  | U.S.         | Corn            | 7.4                                     | 9                      |   |
|  | U.S.         | Corn            | 0.1-25 <sup>d</sup>                     | 21                     |   |
|  | U.S.         | Corn            | trace-25 <sup>d</sup>                   | 2, 21                  |   |
|  | U.S.         | Corn            | 1.1-10.7                                | 26                     |   |
|  | U.S.         | Corn            | 41                                      | 25                     |   |
|  | U.S.         | Corn            | 1.0 <sup>b</sup>                        | 17                     |   |
|  | U.S.         | Oats            | 5                                       | 17                     |   |
|  | Japan        | Barley          | ND                                      | 18                     |   |
|  | U.S.         | Corn            | 1.0 <sup>b</sup>                        | 13                     |   |
|  | U.S.         | Corn            | 0.06 <sup>b</sup>                       | 13                     |   |
|  | U.S.         | Mixed feed      | 0.07 <sup>b</sup>                       | 13                     |   |
|  | France       | Corn            | 0.6 <sup>b</sup>                        | 10                     |   |
|  | South Africa | Corn            | 2.5                                     | 11                     |   |
|  | Zambia       | Corn            | 7.4                                     | 11                     |   |
|  | U.S.         | Corn            | ND                                      | 2                      |   |
|  | Japan        | Barley          | 7.3                                     | 18                     |   |
|  | Austria      | Corn            | 1.3                                     | 24                     |   |
|  | Austria      | Corn            | 7.9                                     | 24                     |   |
|  | Canada       | Corn            | 7.9                                     | 24                     |   |
| Nivalenol  | Japan        | Barley          | ND                                      | 18                     |   |
|  | France       | Corn            | 4.3 <sup>b</sup>                        | 10                     |   |
| Partially<br>characterized<br>trichothecenes           | U.S.         | Corn            | ND                                      | 25                     |   |
|  | India        | Safflower seed  | ND <sup>d</sup>                         | 6                      |   |
| Skin irritant<br>factors—not<br>analyzed<br>chemically | U.S.         | Corn            | 93 positive <sup>b</sup><br>of 173      | 3                      |   |
|  | U.S.         | Corn            | Multiple positive<br>samples            | 21                     |   |
|  | Yugoslavia   | Corn            | 16 positive<br>of 191                   | 1                      |   |

outbreaks that occurred in the Soviet Union. The effects produced are similar to radiation poisoning, and there is a latent phase similar to that seen in radiation poisoning, in which the overt symptoms disappear.

The clinical picture may be divided into four stages.

**The first stage** occurs within minutes to hours after ingestion of toxic grains. The symptomatology described was produced by oral exposure to low doses. In exposure by inhalation, the symptoms may be more pronounced or the time course accelerated. The characteristics of the first stage include primary changes, with local symptoms, in the buccal cavity and gastrointestinal tract. Shortly after ingestion of toxic grain, the patient experiences a burning sensation in the mouth, tongue, throat, palate, esophagus, and stomach as a result of the toxin's effect on the mucous membranes. The tongue may feel swollen and stiff, and the mucosa of the oral cavity may be hyperemic. Inflammation of the gastric and intestinal mucosa occurs, along with vomiting, diarrhea, and abdominal pain. In most cases excessive salivation, headache, dizziness, weakness, fatigue, and tachycardia accompany the initial stage. There may be fever and sweating, but

the body temperature normally does not rise. The leukocyte count may begin to decrease in this stage, and there may be an increased erythrocyte sedimentation rate. This first stage may last from 3 to 9 days.

**The second stage** is often called the latent stage or incubation period because the patient feels well and is capable of normal activity. It is also called the leukopenic stage because its main features are disturbances in the bone marrow and the hematopoietic system, characterized by a progressive leukopenia and granulopenia and a relative lymphocytosis. In addition, anemia and a decrease in erythrocytes, in the platelet count, and in hemoglobin occur. Disturbances in the central nervous system and autonomic nervous systems may occur as well as weakness, vertigo, fatigue, headache, palpitations, and mild asthmatic conditions. Visible hemorrhagic spots (petechiae) begin to appear on the skin, marking the transition to the third phase. The second stage may last 3-4 weeks. The transition to the third stage is sudden, and symptoms progress rapidly.

**In the third stage**, petechial hemorrhages occur on the skin of the trunk, arms, thighs, face, and head. They can vary from a millimeter to a few centimeters in size. Capillaries are fragile, and any slight trauma results in hemor-

rhage. Hemorrhages of the mucous membranes of the mouth, tongue, soft palate, and tonsils occur. Nasal, gastric, and intestinal hemorrhages can be severe. Areas of necrosis begin to appear on the lips, fingers, nose, jaws, eyes, and in the mouth. Lymph nodes are frequently enlarged, and the adjoining connective tissue can become so edematous that the patient has difficulty opening his mouth. Blood abnormalities previously described are intensified. Death may occur from hemorrhage, strangulation due to swelling, or secondary infection.

**The fourth stage** is convalescence. Three or 4 weeks of treatment are required for disappearance of necrotic lesions and hemorrhagic effects. Two months or more may elapse before the bloodforming capability of the bone marrow returns to normal. ■

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